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For Contents See Page 1.

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THE
LARYNGOSCOPE.

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ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

SOME OF THE NEWER METHODS OF THE X-RAY EXAMINATION OF THE PARANASAL SINUSES, THE OPTIC CANALS,* THE PHARYNX AND LARYNX.**

DR. HARRY A. GOALWIN, New York.

Sinuses. The X-ray examination of the paranasal sinuses is probably the most widely used laboratory procedure in rhinology. The rhinologist is also called upon to make his own interpretation of the Roentgenograms more frequently than any other specialist who deals with the diseases of the head. He is therefore interested not only in the finished Roentgenogram, but, to a considerable extent, also in the procedures of making it.

There is a widely prevalent practice of making a diagnosis on sinuses from one or two Roentgenograms. This may lead to serious error even in acute cases, but is absolutely unreliable in chronic cases.

Chronic Sinusitis. A great proportion of the cases of chronic involvement of the sinuses present themselves for the first time to the ophthalmologist rather than to the rhinologist, because they suffer from symptoms referred to the eye rather than to the nose: a drawing sensation in the eye; pain over the eye; pain under the eye; recurrent attacks of conjunctivitis, keratitis, iritis; complicated cataract; floating opacities in the vitreous; retrobulbar neuritis; optic neuritis; secondary infection after eye operations, and other similar complaints.

*The author's researches on the optic canal are part of his work as DuBois Fellow of Columbia University for 1923-1924.

**Read before the Section of Laryngology and Rhinology, New York Academy of Medicine, Nov. 25, 1925.

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In such cases it is necessary to make not only a complete X-ray examination of the sinuses, but also a complete examination of the orbits and optic canals. In other words, one has to consider the sinuses, so to speak, as *paraorbital* as well as paranasal.

We have developed for this purpose a standard technique which gives us sufficient evidence for the examination of all the sinuses, both orbits and optic canals, and a general survey of the entire skull.

Before going into the discussion of this technique and demonstrating some of its results several points should be discussed which are of interest in the interpretation of any film.

1. *Asymmetrical Films.* You frequently have before you a Roentgenogram which shows a difference in illumination between a sinus

Fig. 1. Caudo-dorso-ventral view, illustrating Case 4. (a) Normal left sphenomaxillary fissure. (b) Cloudy right sphenomaxillary fissure, with spicules of bone projecting from maxillary margin. (c) Upper outline of petrous pyramid. (d) Dorsum sellae. (e) Anterior clinoid process. (f) Sphenoidal sinuses.

of one side and the corresponding sinus of the other side, which difference you have to attribute to asymmetrical placement of the head. Merely careful placement of the head will not eliminate such Roentgenograms. It requires actual *measurement* of the relation of the head to the plate. For this purpose the horizontal and median planes of the head are used, as is the practice in anthropology. The soft muslin cap which the author has devised for this purpose is fitted on the patient's head and then the median and horizontal lines of *that* head are marked on the cap. This gives us definite planes, the inclination of which to the plane of the X-ray film can be *measured* by means of a protractor. The procedure enables us to attain a degree of accuracy which warrants considering an asymmetry shown on the Roentgenogram as an asymmetry of the skull; not as technical error.

2. Relative Illumination. A difference of opinion often arises as to the relative illumination of two spots on a Roentgenogram, such as two sinuses of opposite sides of the head. Authority should never be a criterion in the settling of such a dispute, since illumination is a measurable quantity. There are several simple pocket instruments on the market by means of which relative illumination can be measured. Such instruments are: Heyde's actino-photometer and Shepard's density meter.

3. Relation of Illumination to Depth of Sinus, Wall Thickness, etc. When examining a sinus Roentgenogram the first point to consider is the normal illumination which we should expect in the particular sinus investigated. Before striking the film or plate the X-rays (in the posteroanterior views) have to penetrate the pos-

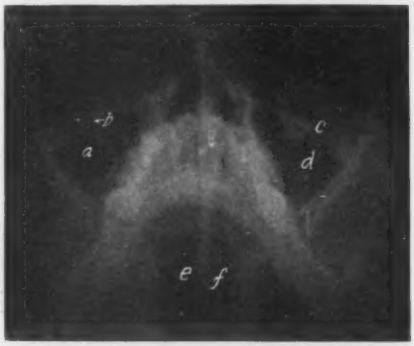

Fig. 2. Axial view, illustrating Case 4. (a) Left maxillary sinus. (b) Left infraorbital foramen: (c) Right infraorbital foramen, with thickened walls. (d) Right infraorbital groove, with thickened walls. (e) Left sphenoidal sinus. (f) Right sphenoidal sinus.

terior wall of the skull, the brain, the posterior wall of the sinus, the cavity of the sinus and the anterior wall of the sinus. If we neglect the brain tissue, the amount of illumination to be expected in a normal sinus depends on the thickness of the bony walls mentioned and the depth of the sinus cavity as measured along the path of the rays. If we take as an arbitrary standard (K) the illumination of a normal sinus 1 c.m. deep, with a thickness of 1 m.m. of the anterior and posterior sinus walls and a thickness of 5 m.m. of the posterior cranial wall, then,

$$Ph=K\frac{t_1+t_2+T}{D} \text{ or } \frac{K}{D}\Sigma t,$$

where Ph is the photometric value of the sinus. t, t and T are the photometric values of the posterior and anterior walls of the sinus

and of the posterior cranial wall, respectively; each expressed in terms of the thickness and density of the standard. D is the depth of the sinus in centimeters.

From this formula we see that the relative illumination of a normal sinus varies directly as the thicknesses and densities of its walls and of the posterior cranial wall, and inversely as the depth of the sinus. The degree of darkness varies therefore directly as the depth of the sinus and inversely as the summation of the thicknesses and densities of the walls mentioned. The indispensability of a *full* lateral view Roentgenogram to reliable diagnosis at once follows, for an apparent clouding may be produced in a sinus by hyperostosis of the posterior cranial wall, of the sinus walls, or both. How a

Fig. 3. Axial view, illustrating Case 5. (a) Left maxillary sinus. (b) Outer wall of orbit. (c) Narrow passage leading into left sphenoethmoidal recess. (d) Sphenoethmoidal recesses. (e) Left sphenoidal sinus. (f) Right sphenoidal sinus.

wrong diagnosis of "unilateral hypertrophic sphenoiditis" was thus made in a case of partial hyperostosis of the sphenoid will be shown in a case cited below (Case 13). How easily a similar error could be made in another case where clouding of one sphenoid was produced by compression of the sinus due to a unilateral tumor in the sella turcica, will also be shown below (Case 10).

4. Uniform and Graded Clouding. The next point to consider is the type of clouding, and whether the bony walls are involved. In acute cases the clouding is uniform and the walls not usually involved. In the early chronic cases, when the mucous membrane is thickened, we see frequently a clear or semiclear area at the center

with a shadow of gradually inceasing intensity as we approach the bony wall. Later the bone also becomes involved. In the maxillary sinus such bone involvement may extend to the infraorbital foramen or groove, or both, thus producing symptoms of an infraorbital neuralgia (Case 8), with pain referred to the various branches of distribution of the superior maxillary division of the fifth nerve. Involvement of the posterior maxillary wall or of the area of the sphenomaxillary fissure produces symptoms referred to the sphenopalatine ganglion (see Case 4).

The idea that the clouding of an inflamed sinus seen on the Roentgenogram is never cleared up after treatment or operation is, if we can judge from our limited experience, a myth. The safest

Fig. 4. Postero-anterior view, illustrating Case 6. Note good illumination of ethmoid cells and total destruction of cell septa.

attitude to assume when such cases are to undergo an operation upon an eye is that *as long as there is X-ray evidence of clouding which cannot be attributed to permanent thickening of the wall, there is some infection present.* When again and again such cases become secondarily infected after an eye operation, one cannot help but feel that great caution should be exercised before considering such cases as cured. On the other hand, in a case cited below (see Case 8), a chronic maxillary sinusitis with Roentgenologically demonstrated periostitis of the infraorbital groove showed complete disappearance of the clouding, of the wall thickening and of the periostitis of the infraorbital groove eleven months after successful treatment.

Ethmoid Cells. In the examination of the ethmoids the structure of the cell septa and evidence of their partial or complete destruction should be noted. A normal ethmoid should show good illumi-

nation and clear-cut septa. An acute ethmoid will show clouding with or without septal obscuration. A chronic ethmoiditis will show septal thickening, decalcification, destruction (case 6); depending on the duration and severity of the condition. Clouding is almost

Fig. 5. Full lateral view (sinistro-dextral), illustrating Case 10.

invariably absent when the cell septa are destroyed (Fig. 4). When present it should lead to a suspicion of tumor rather than inflammation and further evidence of tumor should be sought. Such evidence consists of invasion of the wall, bulging of one or more walls, change in size or contour, or all three.

Sphenoid Sinuses. In the examination of the sphenoids the exact size and shape of the sinus of each side should be noted, and the degree of asymmetry. No examination of the sphenoids is therefore of any value without the axial view (see Figs. 2 and 3). The

Fig. 6. Full postero-anterior view, illustrating Case 10. Note (at c) compression of left sphenoidal sinus downward and mesad.

relation of the sphenoidal sinuses to the optic canals should also be noted, and the size of each optic canal, as well as its shape, determined. If an operation is contemplated the distances of the various walls from the anterior nasal spine should be calculated. They can-

not be taken as measured from the Roentgenogram, because the Roentgenographic measurement may exceed the actual by 15 to 25 per cent. If "a" is half the width of the skull "F"—the focus-plate distance "D," the Roentgenographic distance and "x," the actual distance, then,

$$x = D\,\frac{F-a}{F}$$

If the Bucky diaphragm is used and "b" is the distance from the surface of the diaphragm to the surface of the plate or film, then this formula becomes,

$$x = D\,\frac{F-(a+b)}{F}$$

Frontal Sinuses. As the frontal sinus is the most variable in depth, its anteroposterior dimension should be particularly noted

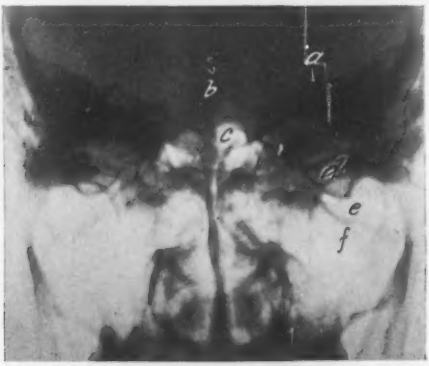

Fig. 7. Caudo-dorso-ventral view, illustrating Case 10. Note clouding of left sphenoidal sinus. (a) Upper outline of petrous pyramid. (b) Dorsum sellae. (c) Right sphenoidal sinus. (d) Orbital surface of greater wing of sphenoid. (e) Sphenomaxillary fissure. (f) Maxillary margin of sphenomaxillary fissure.

before any judgment is passed upon its illumination. What is normal illumination for a sinus 2 m.m. deep is certainly pathological for a sinus 2 c.m deep, other factors remaining the same. Absence of a frontal sinus should not be diagnosed until such absence can be demonstrated on the oblique view used for the optic canal. If this rule were followed the diagnosis of absence of a frontal sinus would be made with much less frequency than it is made now. It would indeed be a rare occurrence. The differentiation between absence and obscuration of a frontal sinus can be made without difficulty from a properly complete set of Roentgenograms, such as will be described below.

"Spongiozation of Frontal Sinus. In this connection it may be well to mention a comparatively little known condition of the frontal sinuses which was first described by Preysing,[15] in 1910, as "Spongiosierung des Sinus frontalis." He described this as a condition characterized anatomically by filling of the frontal sinus with a highly vascular, spongy tissue similar to that which is sometimes found in chronically inflamed mostoids before the stage of sclerosis. Clinically it is characterized by fairly severe headache and pain over the affected side, but without tenderness. Roentgenologically there is an apparent absence of the sinus, but a faint wall can be made out. The characteristic inflammatory area which is found around an obscured infected sinus is, however, absent. He reported at that time eight cases, in all of which the etiology was obscure. In no case could he find any evidence or history of previous inflammation of the sinus or any relation to any general disease. All his patients

Fig. 8. Cephalo-dorso-ventral view, illustrating Case 13. Note thickening and increased density of both margins of the sphenoidal fissure of the right side.

were females. He believes that it may be due to a circulatory disturbance, because most of them dated the beginning of their symptoms back to puberty. One of his cases also had hyperostoses in the frontal sinus affected; one case had also middle ear sclerosis; and his last case had also a purulent maxillary sinusitis on the affected side. He operated on two of these cases and found the pathology described.

Tuberculosis of Sphenoid. Tuberculosis of the sphenoid was demonstrated on the Roentgenogram by Schüller three times. The first two cases have been proven by autopsy. We saw the third of these cases on his service (Zentral Roentgen-Institute des allgemeinen Krankenhauses in Wien-Vorstand Prof. Holtzknecht), in 1924. This patient was sent for X-ray examination from the eye clinic

with a diagnosis of retrobulbar neuritis. All three cases had pulmonary tuberculosis. The condition is demonstrated on the lateral Roentgenogram as usually taken for the sella turcica. The picture is characteristic: One notices at first sight an apparent destruction of the entire body of the sphenoid, as in advanced cases of tumor of the pituitary. Closer inspection reveals a phantom-like *normal* outline of the sella turcica and a mottled, decalcified sphenoidal body. According to Schüller[2] the disease begins by infection of the sphenoidal sinuses from the tuberculous material which the patient coughs up into the nasopharynx. The infection of the mucous membrane which this produces, cannot, of course, be at first differentiated from any other kind of infection of the sphenoidal sinuses. It is only when the bone becomes involved that the typical Roentgenographic picture can be seen.

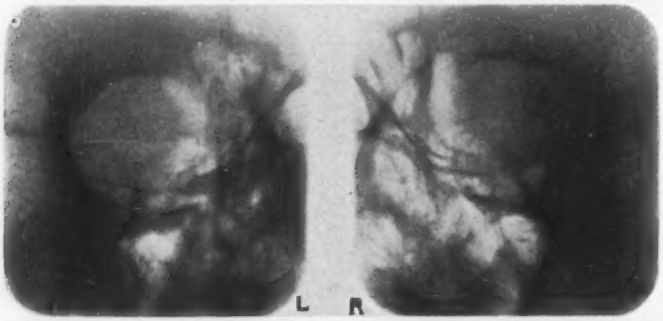

Fig. 9. Right and left optic canal Roentgenograms, illustrating Case 13. Note marked hyperostosis of apex of right orbit, and triangular area of lesser density at floor of right optic canal. Note normal left optic canal.

Complete Examination of Sinuses. From the foregoing discussion it is evident that for the complete examination of all the sinuses a full lateral (Fig. 5), a full posteroanterior (Fig. 6), an axial (Figs. 2 and 3), and two optic canal Roentgenograms (Figs. 9, 10, 11), are required. In addition to these, two other Roentgenograms are necessary, the caudodorsoventral (Figs. 1 and 7), and the cephalodorsoventral (Fig. 8).

The lateral Roentgenogram (Fig. 5) gives us the thickness of the skull and the thicknesses of the various sinus walls, the depths of the sinuses and their relations to each other, to the orbits and the optic canals. It gives us, furthermore, a lateral view of the orbits, the distance of the sphenoids from the anterior nasal spine, indicates the presence or absence of sphenoethmoidal or frontoethmoidal

recesses, or of supraorbital recesses in the frontal sinuses. It gives us also a lateral view of the sella turcica.

The straight posteroanterior view (Fig. 6) shows all the sinuses, including even the sphenoids when these extend under the sella turcica. It shows also the thicknesses of the lateral walls of the sinuses and of the skull, the orbital margin and the sphenoidal fissure. As the shadow of the petrous pyramid covers in this view the upper third of the maxillary sinus and the lower third of the orbit, we have to resort to the next described view, in order to project the maxillary sinuses and orbits free from the pyramids.

The cephalodorsoventral (Fig. 8) Roentgenogram shows the same structures as the straight posteroanterior view, but the maxillary sinus is not covered by the petrous pyramid. It gives us also a pro-

Fig. 10. Illustrating Case 13. Note distortion and apparent constriction of right optic canal, due to faulty position. Compare with correct Roentgenogram of right optic canal of same case in Fig. 9.

jection of the orbital roof into the area enclosed by the orbital margin.

In the caudodorsoventral view (Figs. 1 and 7) the sphenoidal sinuses are projected over or above the frontals, depending on the vertical extent of the latter. The sphenoids appear as an oval-shaped structure of which the long axis is horizontal. Below each apex of the oval are the posterior ethmoid cells. If the other views show clouding of the frontals great caution should be exercised in deciding upon the superimposed sphenoids. Similar caution should be exercised when the sphenoids are projected over frontals which show unequal illumination due to unequal depth.

The caudodorsoventral Roentgenogram also gives an excellent view of the sphenomaxillary fissure. In some cases of maxillary or

retromaxillary infection hyperostoses or exostoses of one or both margins of this fissure may be seen. Projected above the sphenoidal sinuses one sees in this Roentgenogram the anterior and posterior clinoid processes and the upper outline of the petrous pyramids.

The axial view (verticomental) (Figs. 2 and 3) shows the relation between the sphenoidal sinuses of the right and left side, the maxillary sinuses, an outline of the inner and outer walls of the orbits, the infraorbital foramen and, sometimes, the infraorbital groove. A frontal sinus which appeared to be absent in the previous views may show up in this Roentgenogram. While this view is not needed for the determination of pathology in the maxillary sinuses it is of definite help when such pathology was doubtful in the other views, or when the infraorbital foramen or groove is involved.

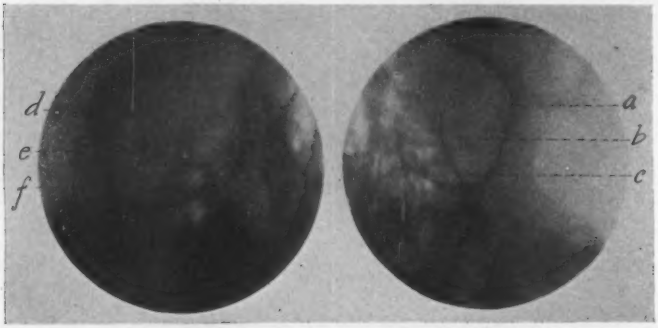

Fig. 11. Optic canal Roentgenograms, illustrating Case 12. (a) Orbital margin of right side. (b) Normal right optic canal. (c) Normal wall of right sphenoidal sinus. (d) Constricted left optic canal with hazy wall outline. (e) Pneumatized, sclerosed left anterior clinoid process. (f) Thickened walls of left sphenoidal sinus.

Sphenoethmoidal recesses which are situated lateral to the sphenoidal sinuses are seen on this Roentgenogram, while those situated above or below the sphenoidal sinuses are seen in the lateral view.

Optic Canals. The optic canal Roentgenograms (Figs. 9, 10, 11) show, in addition to the shape, size and wall structure of the optic canals, also the orbital margin and oblique views of the sphenoid, ethmoid, maxillary and frontal sinuses. This Roentgenogram will show up a frontal sinus no matter how rudimentary that sinus may be, and even when all the other Roentgenograms described failed to demonstrate its presence.

The X-ray examination of the optic canals is of interest to the rhinologist, primarily because of their relation to the sphenoids or

posterior ethmoids. It was from this point of view that the study of the optic canals was made by White.[7] We have been able to confirm his conclusion[12] that when a unilateral optic atrophy follows as a result of a bilateral sphenoiditis, it is the side with the smaller of the two canals that is involved, provided, however, that each canal is related in the same degree with the sphenoid of its side. As regards to flattening of the optic canal walls wherever there is excessive pneumatization, our findings do not agree, except when the anterior clinoid process is pneumatized. In that event the corresponding wall of the optic canal is more than flattened; it actually bulges into the lumen of the canal. Whenever inflammation and

Fig. 12. Roentgenograms of normal pharynx. (a) Nasopharynx. (b) Soft palate. (c) Uvula. (d) Base of tongue. (e) Pharynx. (f) Cervical vertebrae.

subsequent sclerosis can be demonstrated in such a pneumatized anterior clinoid process there is an associated hyperostosis or exostosis of the optic canal wall and a constriction of its lumen (Fig. 11). Optic atrophy is naturally present under such circumstances and the prognosis at this stage is hopeless.

For precise measurement the technique we have described[9 10 11] should be followed. Our average as determined by numerous measurements[8] is much lower than that given by White, probably because his figures relate to the Roentgenographic diameters, while ours are reduced by calculation, so that they represent the *actual* size.

Our average diameter of 4.3 m.m. for the cross-section of the optic canal as determined from measurements on 200 optic canals in the skeleton, is followed very closely in our calculations of 400 optic canals in the living.[22]

The rhinologist should be familiar also with some ophthalmologic phases of the Roentgenography of the optic canal because the optic canal Roentgenogram is in some cases the only means of differential diagnosis between an exophthalmos or optic nerve atrophy produced by sinusitis and a similar condition produced by an orbital or optic nerve tumor.[12 13] The diagnostic principles and other data on optic canal Roentgenography are covered in the works of Schüller,[1] de Kleyn and Stenvers,[4] Herrnheiser,[6] van der Hoeve,[5] White,[7] Goalwin,[8 9 10 11 12 13 22] and others. These authors have reported fractures

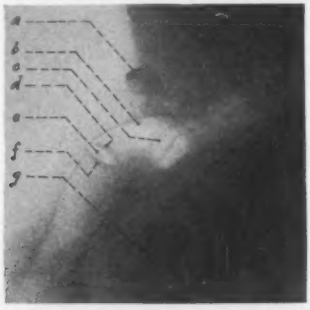

Fig. 13. Roentgenogram of normal larynx. (a) Hyoid bone. (b) Epiglottis. (c) Aryteno-epiglottidian fold. (d) Superior vocal chords. (e) Ventricle. (f) Inferior vocal chords. (g) Cervical vertebrae.

hyperostoses, exostoses, periostitis, constriction and enlargement of the optic canal, tumors, changes in shape due to excessive pneumatization of the adjacent structures (White, Goalwin) and changes in shape and size associated with skull deformities (Goalwin).

As we have pointed out in our previous writings,[9 12] the size of the cross-section of the optic canal cannot be taken directly from the Roentgenogram. The size of the focal spot of the tube used should be measured. The distance of the focal spot from the tube should be measured each time. The distance of the optic canal from the lower outer margin of the orbit can be taken from our curves.[8] These curves, which are based on measurements of a large number of skulls, give the average distance for any age, beginning at birth. To this distance should be added the distance from the lower outer angle of the orbit to the plate.

If x be the actual diameter of the cross-section of an optic canal,
d, the Roentgenographic diameter corresponding to it,
a, the distance from the focal spot to the plate,
b, the distance from the optic canal to the plate, and
f, the diameter of the focal spot of the tube used,

Then $$x=d-\frac{a}{b}(f+d)$$

Pharynx and Larynx. The pharynx and larynx are not frequent objects of X-ray examination. The reason for this is, of course, that these structures are accessible to instrumental examination. Yet the X-ray examination of the pharynx and larynx can be of material aid to the clinician. The high air content of these structures makes many pathological processes in them easily demonstrable on a properly taken Roentgenogram. The profile view is practically the only one we can use because of the superimposition of the vertebrae on any other view. For the pharynx the tube is centered over the angle of the mandible. For the larynx it is centered over the thyroid cartilage, as closely as possible over its center.

A good Roentgenogram of the pharynx (see Fig. 12) shows the nasopharynx, the soft palate, the base of the tongue, the cervical vertebrae and the uppor portion of the larynx. Adenoids, tumors, tuberculous spondilitis, stricture, etc., can readily be demonstrated. Thost demonstrated exostoses of the anterior walls of the cervical vertebrae, calcification of interverebral cartilages, etc., in cases which complained of otherwise obscure pharyngeal symptoms. The presence and exact extent of retropharyngeal abscess is easily demonstrated. When such an abscess extends into the hypopharynx, the larynx, which normally lies in front of the collapsed hypopharynx close to the vertebrae, is pushed forward, giving a very characteristic picture.

Gutzmann, Scheier and Weingartner have made a study of the behavior of the pharynx during the acts of swallowing, speaking and singing, using the methods of fluoroscopy and kinematography for this purpose.

Unusually marked lordosis of the cervical spine may upon instrumental examination be mistaken for swelling. Such a mistake is impossible in the X-ray examination. Unusually long styloid processes have been demonstrated by von Eicken[18] in cases where obscure difficulty of swallowing existed. The exact topography of fistulae can be demonstrated by filling with an opaque paste or with lipiodol, in a manner similar to that of demonstrating the lacrymal ducts and some of their pathology.

In order to understand the possibilities and limitations of the Roentgenography of the larynx one must be familiar with the normal process of ossification which goes on in its cartilages throughout life. This has been described in detail by Scheier and Fränkel. According to these authors ossification here begins in the male at about the age of 18 years, and in the female at about 15. In the thyroid cartilage it begins in both sexes at the posteroinferior angle. In the male other ossification centers develop later, the ossification continuing along the margins, so that at the end of the fourth decade we find as a rule the entire thyroid cartilage enclosed in a bony frame. There is, however, a narrow strip of bone extending upward and forward from the tuberculum inferior, almost to the superior margin. By extension this bony framework gradually covers the entire thyroid cartilage. In the female no other ossification center develops. The ossification extends upward along the posterior margin and then forward. The forward progression is either uniform or staircase-like. Total ossification never takes place in the female thyroid cartilage.

It is evident, therefore, that when the ossification of the thyroid cartilage is complete the details of the soft structures of the larynx are no longer demonstrable on the X-ray picture, that the best results can be expected in children, in women, and in men under 50.

Thost[19 20] finds arrest or retrogression of the normal ossification process in cases of tuberculosis; increased ossification with indentations in cases of syphilis; increase, alternating with rarefaction, in cases of carcinoma.

The profile Roentgenogram (see Fig. 13) of the normal larynx shows very clearly the ventricle, the superior and inferior vocal cords, the epiglottis and the arytenoepiglottidian fold. Stenosis, foreign bodies, edema, tumors, nodules, etc., are easily demonstrable. Polypi in the ventricle can be easily seen even when very small. The exact origin of pedicled tumors can be determined. The X-ray method of examination is particularly valuable in children, in whom instrumental examination is difficult, and in cases where swelling or stenosis obstructs the view from above.

A posteroanterior Roentgenogram of the larynx is obtained by Rheti,[17] who introduces for this purpose a small film into the cocainized hypopharynx. This method gives the finest normal and pathological details.

The following case histories, together with the X-ray reports and Roentgenograms, are intended to illustrate the principles and clinical value of the type of Roentgenography which we advocate in this article:

CLINICAL ILLUSTRATIONS.

Case 1: R. E., age 32 years, has had several operations on frontal sinuses during the past seven years, without relief from his symptoms. X-ray examination shows: Both frontal sinuses multilocular, the left consisting of five pockets, four vertical and one horizontal, the horizontal compartment covering about one-half of the orbital floor. The right frontal sinus consists of three vertical pockets and one horizontal extension covering half of the orbital floor.

The left frontal sinus is normally illuminated. In the right frontal sinus the middle pocket is separated from the main cavity by a horizontal septum, leaving only a small communication about 6 m.m. in diameter. This middle pocket is markedly cloudy, the remainder of the sinus being clear. The anatomy of this sinus should be carefully noted before any operative procedure is undertaken.

Case 2: P. M., age 56 years, cataract extraction right eye ten years ago, followed by unexplained optic atrophy. Cataract extraction left eye four years ago, followed by optic atrophy.

X-ray examination shows constriction of left optic canal to 2.8 m.m., which constriction is produced by bulging of a pneumatized anterior clinoid process into the lumen of the canal. There is evidence of chronic bilateral sphenoiditis and posterior ethmoiditis. There is thinning of the upper and lower margins of the left sphenoidal fissure with obscuration of its outline; and thickening of the anterior part of the left orbital floor. We are evidently dealing with an old infection of the sphenoids and posterior ethmoids, and with an infection of the left orbit, the constriction of the left optic canal being a result of the former process.

Case 3: F. W., age 22 years. Chronic blepharoconjunctivitis with acute exacerbations several times a year. Duration, four years. X-ray examination shows evidence of bilateral frontal sinusitis. Operation revealed purulent sinusitis. Lids and conjunctiva have remained clear to the present time (eight months).

Case 4: L. N., age 35 years, complains of sensation of "tugging and pulling" on the right eye, during the past four months. X-ray examination was made one week ago and reported negative.

X-ray examination shows moderate clouding of the right maxillary sinus. The sphenomaxillary fissure shows narrowing and clouding, and small spicules of bone projecting from the maxillary margin into the fissure. There is some thickening of the wall of the right infraorbital foramen and groove. *This is a chronic right maxillary sinusitis with involvement of the sphenopalatine and sphenomaxillary regions, and of the infraorbital foramen and groove.*

Case 5: A. C., age 36. Marked reduction of vision during past fifteen days. Now has papilloedema and central scotoma, o. u.

X-ray examination shows: The sphenoidal sinuses extend under the sella turcica. On either side of these is a recess which communicates by a narrow passage about 2 c.m. long with the ethmoidal area. Thees are sphenoethmoidal recesses. They show evidence of inflammation. After operation the eyes cleared up and vision returned to 20/20 within one week.

Case 6: I. R., age 60 years. Iridocyclitis following cataract extraction. X-ray examination shows well illuminated ethmoid sinuses with total destruction of septa on both sides. X-ray diagnosis: chronic bilateral ethmoiditis. Nasal examination shows purulent discharge from ethmoidal area.

Case 7: T. N., age 50 years. Sticking pain and exophthalmos, left, two months. X-ray examination shows evidence of left chronic ethmoiditis and left maxillary sinusitis.

Case 8: Dr. M. B., infraorbital pain several years, left side. X-ray examination shows evidence of left maxillary sinusitis with clouding of infraorbital foramen and thickening and haziness of its wall.

On operation a small amount of mucopurulent material was washed out of this sinus. Washing repeated several times. Eleven months later patient reported for another X-ray examination. He was then free of all symptoms. The sinus and infraorbital foramen have cleared up.

Case 9: F. O., age 20 years. Right retrobulbar neuritis for which the right sphenoid had been operated, without relief. Now has beginning involvement of left eye.

X-ray examination shows, in the axial view, that the left sphenoidal sinus is L-shaped and extends behind the right sinus, reaching to the right optic canal. We introduced a probe into the right sphenoid and made Roentgenograms (lateral and axial) with the probe in situ, to further demonstrate the anatomical variation. Under treatment of the left sphenoid the ocular symptoms subsided in both eyes.

Case 10: I. S., age 45 years, had cold four months ago. Occipital pain since then. Had several colds since. Pain always returns with cold. Gradually diminishing vision in right eye about twelve years. Diminishing vision in left eye noticed lately

X-ray examination shows: Enlargement of the sella turciea, which enlargement is only moderate. The floor shows a slight double contour. In the posteroanterior view, however, the body of the

sphenoid is seen to be compressed from the left downward and inward. In the caudodorsoventral view the left sphenoidal sinus appears cloudy, but this is due not to sphenoiditis, but to the reduction of the height of the sinus on this side. There is some separation of all the sutures and enlargement of all the pneumatic spaces. The optic canals are normal in shape, size and structure of their walls. Their calculated diameters are: Right, 3.9x4.7 m.m.; left, 3.9x4.7 m.m. *We are dealing with a lesion of the pituitary, which is situated mainly on the left side.*

We were able to say from the X-ray examination alone that the patient should have a right homonymous hemianopsia, which statement was borne out by the clinical examination.

Case 11: E. B., clinically optic atrophy (secondary) left. X-ray examination shows: Right optic canal normal. Left optic canal constricted, its lower outer wall thickened, and a spur extending from it to the center of the lumen. The adjacent anterior clinoid process is pneumatized, the walls of the cavity being increased in thickness and density. The thickening and exostosis are due to chronic inflammation within this anterior clinoid process.

This case, as well as the next one, is cited because it shows the enormous value of the optic canal Roentgenogram in establishing the prognosis in such cases.

It is important to note that these are not instances of optic atrophy due to small canals in presence of sphenoiditis. These optic canals were constricted by the periostitis which resulted from the sinus infection. Whether the optic nerves were involved before this process of constriction began, whether they were originally smaller than normal, etc., cannot be told at this stage. Unilateral retrobulbar neuritis does occur in the presence of unilateral sphenoiditis in cases where the optic canal of the affected side has a relatively large cross-section. In our experience such cases of unilateral retrobulbar neuritis are much more frequent than cases of bilateral sphenoiditis with unilateral involvement of the optic nerve, due to its smaller size, as compared with the opposite side.

Case 12: H. P., recent impairment of vision, right. Secondary optic atrophy, left.

X-ray examination shows: Right optic canal normal. Left optic canal constricted to 2 m.m. (calculated). Its walls are thick and irregular. The adjoining anterior clinoid process pneumatized and sclerosed. Bilateral chronic sphenoiditis. No evidence of hyperostoses elsewhere in the skull. This is a constriction due to chronic sphenoiditis.

Case 13: E. C., age 49 years, blurred vision in right eye eight months ago, vision 20/70. Was diagnosed six months ago as retrobulbar neuritis, due to right sphenoiditis of "hypertrophic" type, and right sphenoid operated upon.

Her vision now is 4/200. She now has a large central scotoma, swelling of the upper lid, exophthalmos with displacement of the eye outward as well as forward. X-ray examination now shows: There is marked thickening and increased density of the lesser wing of the sphenoid and of the orbital portion of the great wing. The left optic canal is normal in shape, size and structure of its walls. The right optic canal is *larger* than the left. Its shape is normal. Its walls are thickened. They are continuous with the osteomatous area of bone surrounding the canal. Below is a triangular area of decreased density. Above and temporally is a fine, fissure-like erosion. The calculated diameters of the optic canals are: Right, 3.9x5.1; left, 3.7x4.7 m.m. Such slight difference in size cannot be considered pathological. It is the extensive osteomatosis associated with fine erosions which indicates that we are probably dealing with an *endothelioma* of the optic nerve sheath. The process apparently has an intracranial extension, which extension has a small focus of cystic degeneration in its superior portion. The sella turcica is normal in shape and size. The dorsum sellae is normal. The left anterior clinoid process is normal. *The right anterior clinoid process is short and sharp.*

We have gone into a detailed description of this case because, like many other cases of optic tumor, it was at first considered a rhinological case. Because careful Roentgenography may avoid this error in such cases, we feel justified in discussing this case a little further.

There is one incontrovertible fact in this case: We are dealing with a partial hyperostosis of the sphenoid. *Simple hyperostosis* in this region *can only produce eye symptoms* (optic atrophy, vascular congestion, exophthalmos, paralysis of ocular muscles) by *constriction* of the optic canal or sphenoidal fissure. *There is no constriction in this case.* This leaves only two possible causes for the eye symptoms in this case: *1*, orbital infection following sinusitis; *2*, tumor. The lack of clouding of the sphenoidal fissure and absence of evidence of ethmoiditis speak against infection. The venous dilatation seen in the fundus, the fine erositions and the limitation of the bone thickening to the apex of the orbit speak for tumor. Such hyperostoses have been found by Cushing overlying endotheliomata of the dura elsewhere in the skull. Penfield states that they

occur not infrequently in the orbit, and that wherever they occur, trauma cannot be dismissed as the cause.

SUMMARY.

1. The complete X-ray examination of the sinuses requires at least seven Roentgenograms: the lateral, posteroanterior, cephalodorsoventral, caudodorsoventral, axial, right and left optic canals.

2. Each sinus has a normal illumination which depends upon its depth, the density and thickness of its own walls and those of the skull. Before deciding upon the condition of a sinus the normal illumination to be expected should be estimated.

3. To make this estimate possible, a full lateral and full posteroanterior view should be taken.

4. The position of the head should always be *measured* by measuring the inclination of its principal planes to the X-ray plate, using a protractor.

5. In case of doubt the illumination of the sinus should be *measured* by means of some form of photometer.

6. The Roentgenologist should be thoroughly familiar with all the clinical and Roentgenologic aspects of the disease and deformities of the head, and, needless to say, with the finest details of the anatomy of the head.

7. Great precaution is necessary in the Roentgenography of the optic canals. The size of the focal spot of the tube should be measured. The distance of the focal spot from the plate and of the canal from the plate should be noted.

8. The size of the optic canal cannot be taken directly from the film. It must be calculated.

9. The Roentgenologist's duty does not end when he makes a diagnosis. *He should furnish the clinician with all the anatomical data he can determine from his Roentgenograms, which will help in treatment or operation.*

10. The X-ray examination can be of great service to the clinician in the diagnosis of diseases of the pharynx and larynx.

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THE DIAGNOSIS AND TREATMENT OF PARANASAL SINUS INFECTIONS IN INFANTS AND YOUNG CHILDREN UNDER ETHYLENE ANESTHESIA.*

DR. L. W. DEAN, Iowa City, Ia.

The treatment of paranasal sinus suppuration in infants and young children is, as a rule, a very simple procedure. At least 90 per cent of the cases are cured by the removal of tonsils and adenoids with simple toilet of the nose and proper diet. The remaining 10 per cent can only be eradicated by change of climate or by more complicated treatment, which must be done by the laryngologist. It is frequently necessary to wash out the maxillary sinuses and inject some mild antiseptic and astringent in addition to cleansing the ethmoidal region. In our experience it has been the cases associated with severe systemic condition, such as arthritis, chorea, and nephritis, that have been slow to yield to treatment. The systemic conditions are frequently reported by the pediatrist as improved, but not cured. Examination has demonstrated the sinus condition has been much improved, but not completely cured.

The erdication of the last trace of sinus infection has in many instances brought a much more favorable report from the pediatrist. It is only natural to expect that these more chronic forms associated as they are with marked changes in bone and periosteum, inflammatory in nature, are the ones most likely to produce the systemic complications. Naturally this type is more difficult to cure.

We have had little difficulty in diagnosing chronic sinus infection in this class of cases, but our difficulty has been the eradication of the last trace of the sinus disease.

Treating the maxillary sinuses by irrigation and cleansing the ethmoidal regions in infants and young children is unsatisfactory if no anesthesia is used. It is very nerve-rcking. The patient cries throughout the treatment. It struggles and may cause traumatism with some undesirable complications.

Nine months ago we tackled the problem of treating sinuses under general anesthesia. We have devised a fairly satisfactory procedure. For the first time, we have had rapid improvement

*From the Department of Head Specialties, University Hospital.
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in the more difficult cases and have been satisfied with our results.

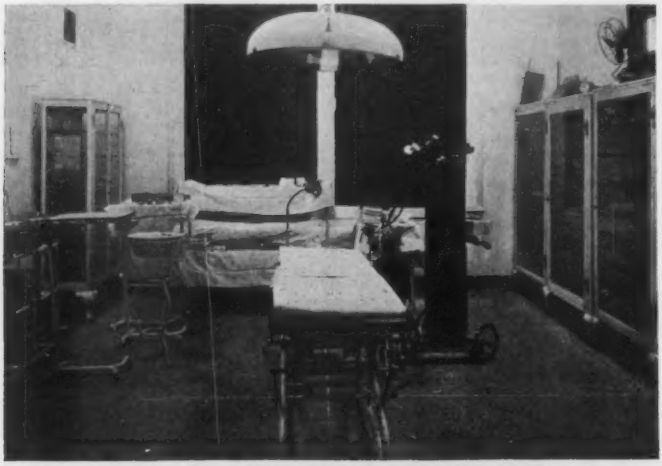

Plate I. Arrangement of the operating room.

Plate II. Mouth piece for administration of ethylene.

Our problem was explained to Dr. L. W. Harding, head of the Department of Anesthesia in the University Hospital, and to Dr. Mary Ross, the anesthetist of the head specialty service.

In succession chloroform, ethyl chlorid, nitrous oxid and oxygen and ethylene were used as the anesthetic. For one reason or

Plate III. Use of nasopharyngoscope under ethylene anesthesia.

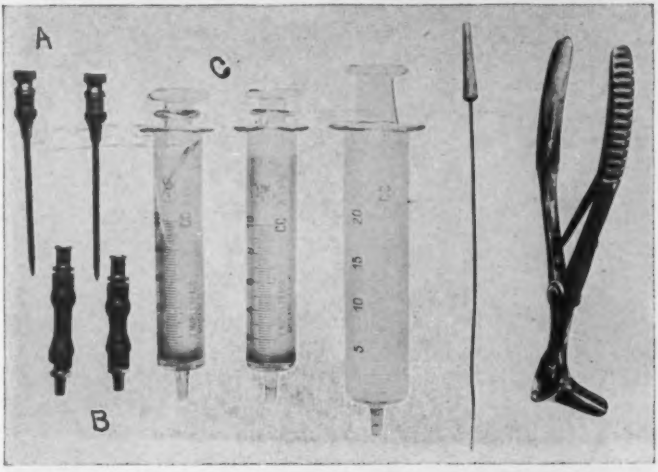

Plate IV. Instruments for studying maxillary sinuses: A. Trochar with needes; B. connecting pieces; C. 10 c.c. Luer syringes for injecting mild antiseptic.

another, all were discarded except the ethylene. Nitrous oxid and oxygen worked nicely for three or four times with an infant

and then the patient seemed to acquire a tolerance for the gas and jerked throughout the treatment.

Plate V. Inserting trochar into maxillary sinus.

Plate VI. Aspirating maxillary sinuses.

Ethylene, when given by mouth by Dr. Ross, for the purpose of treating the sinuses in infants and young children seems to

be the ideal anesthetic. The patients may be treated lying on the back or side. They are perfectly quiet. The anesthetic has no terrors for them. They do not cry before or after the anesthesia. I am informed by Dr. Jeans, Head of Department of Pediatrics, that he has noted no bad results. Even in infants and young children with diabetes, when properly prepared by the pediatrist for the treatment, there has been no deleterious results. I am certain it is necessary to have a very expert anesthetic service to get satisfac-

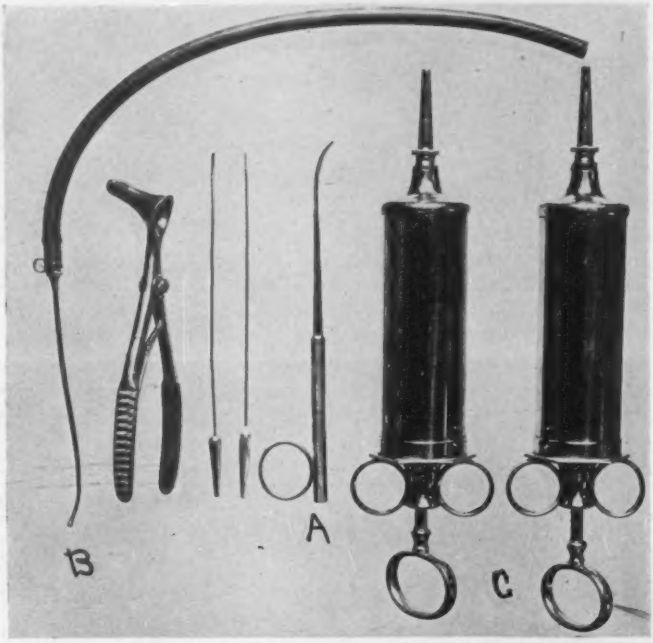

Plate VII. Instruments used in treating maxillary sinus: A. Rasp of infants; B. maxillary sinus canula; C. metal syringes.

tory results. It is very difficult to keep the patient in just the right stage of anesthesia.

The diagnosis of sinus disease in infants and young children may be made nicely with ethylene anesthesia. If operations are performed on the nose or sinuses, Dr. Ross prefers chloroform and oxygen because the field is much less bloody, and electrically-driven suction machines may be used in the operating room in safety.

At present we prefer not to use any machine that may spark in the room where ethylene is being given. We are planning

Plate VIII. Position of patient for treatment of maxillary sinuses under ethylene anesthesia.

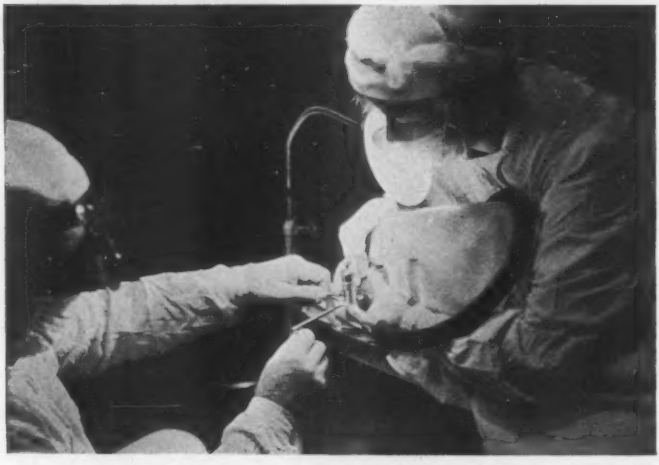

Plate IX. Making the meatal opening in the inferior meatus, using the rasp.

on screening the motors and in this way obviating the danger from sparking and explosion.

DEAN: PARANASAL SINUS INFECTIONS.

In examining the paranasal sinuses in infants and young children for diagnostic purposes, it is advisable to have the patient

Plate X. Canula in maxillary sinus ready for washing.

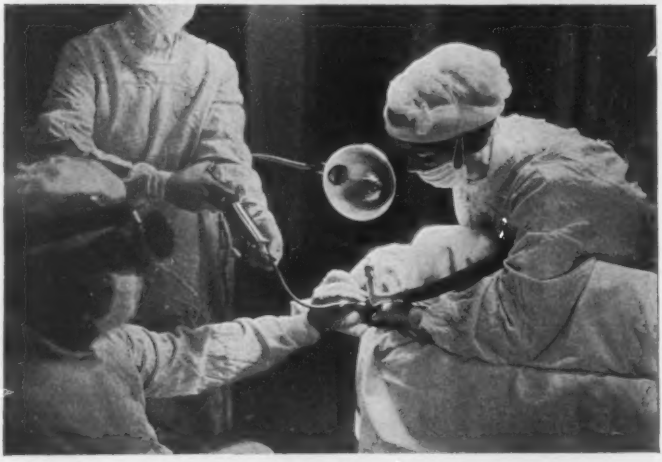

Plate XI. Washing the maxillary sinus.

asleep. Before giving the anesthetic an X-ray examination of the sinuses should be made. The most important information

thus gained is the determination whether or no a given sinus on an anatomical basis is of clinical significance. The blurring of the sinus on the plate or the absence of blurring is of value. The usual examination of the nose for discharge should be made. The presence or absence of cervical adenitis should be noted, the condition of the pharynx determined, and a thorough general examination made before the anesthesia. Under ethylene anesthesia it is easy to determine the presence of adenoids. If present and it is desired to remove them, this can be readily done immediately following the examination with the ethylene anesthesia.

In my work, I find it advisable to have one physician give the anesthesic. It is the only way to get the best teamwork. The operator becomes accustomed to the anesthetist, and vice versa.

The difficulties of anesthetizing are greater in nose and throat work than in any other branch of surgery. It is very necessary that the operator do quickly things that the anesthetist asks him to do.

First, as to the method of diagnosing paranasal sinus disease under ethylene anesthesia.

Plate I shows the equipment of the operating room. To the right is the illuminated box for the X-ray plate. This is so placed that the operator may watch it while operating and be guided by it in entering the maxillary sinuses. To the right of the table is the ethylene tank, the oxygen tank, and the anesthetic apparatus. In our operating room to the left, an emergency tracheotomy set and syringe and needle for injecting the heart with adrenalin are always ready. We have never had use for either in ethylene anesthesia.

Plate II shows the mouth piece in position. Note that the anterior nares are accessible.

Plate III shows the examination of the nose with the nasopharyngoscope. Dr. Ross, giving the anesthetic, is at the head of the table. She also holds the infant's head.

During the last three months in investigating the maxillary sinuses, we have used a new technique. Instead of inserting a long needle through the trochar that has been passed into the sinus, a syringe is attached directly to the trochar. Sterile normal salt is injected into the sinus and aspirated through the trochar. The trochar has an interior diameter three times that of the needle formerly used and larger pieces of pus and thicker pus may be aspirated. It also obviates the danger of injuring

the sinus wall by a second needle, which, as originally used, projected beyond the end of the trochar.

The material aspirated is examined macroscopically for pus and is sent to the laboratory for microscopical examination and culture.

Plate IV shows the instruments used in the study of the maxillary sinuses. To the left are two trochars, one for each sinus. Next are two 10 c.c. Luer syringes. To connect the trochars with the syringes are two flexible tubes. We smear the joints with vaselin to make them airtight. To the right is a 20 c.c. Luer syringe containing argyrol, which may be injected into the sinus for therapeutic purposes if desired at the conclusion of the investigation.

Plate V shows the trochar being inserted into the sinus. We have always passed the trochar through the inferior meatus. Before passing the trochar, the meatus is swabbed with 95 per cent alcohol. If the X-ray plate shows the floor of the maxillary sinus to be above the attachment of the inferior turbinate, the trochar is inserted upwards and outwards through the attachment of the inferior turbinate to the wall of the nose. The floor is frequently above the inferior turbinate in infants.

Plate VI shows the syringe attached to the trochar which has been inserted into the maxillary sinus.

For treating the maxillary sinuses, a small meatal opening is made through the inferior meatus. Through this opening a maxillary canula is inserted for the irrigation of the sinus and the injection into it of mild antiseptics. We use argyrol mercurochrome and ½ per cent silver nitrate. When the latter is used the nose and lips are smeared with vaselin. It is important to remember that in infants and young children, the curve of the sinus canula must be very much less than that necessary for the adult. We mould our canula so it is the same shape as the maxillary sinus rasp.

Plate VII shows the instruments used in treating the maxillary sinuses.

"A" is a rasp for making the meatal opening.

"B" is the maxillary sinus canula, very flexible. Attached to it is a weak rubber tube. The tube is easily distensible and indicates by swelling how much pressure is being applied during the irrigation. The swelling of the tube indicates that the return flow is blocked and that the treatment should be discontinued until this is corrected.

"C" are two metal syringes; one for water used in washing, the other for the injection of the mild antiseptic.

Plate VIII shows the position of the infant for treatment. The patient is on the side so the fluid injected will escape from the anterior nares.

The operator sits on a low stool on the floor. Dr. Ross is behind the patient supporting the head. A nurse holds the infant's hips, the child being doubled like a jack-knife. The light for reflected illumination is above the infant's head in a position convenient for the anesthetist.

Plate IX, the meatal opening is being made with the rasp.

Plate X shows the maxillary canula in position.

Plate XI shows the washing out of the sinus.

Before treatment the nose is cleansed, using cotton on a probe. After the treatment of the maxillary sinuses, the nose is again cleansed and the ethmoidal region carefully brushed with some mild antiseptic.

AN UNUSUAL BREAK OF THE FACE.

Dr. William A. Boyce, Los Angeles, Calif.

A male, age 41 years, while driving at a rapid rate of speed, had a head-on collision with another car.

The impact catapulted him through the windshield into the other car. He was picked up and taken to an emergency hospital, and was found to have a broken right femur, and his nose was bleeding. The break in leg was set and both nostrils packed with gauze. I was called several hours later to see if he had a broken nose. After removing the packing from nostrils, I found that the whole of his face was freely moveable. By grasping the front upper teeth, which had receded back of the lower teeth, the whole superior maxilla could be moved to the side, or backward and forward.

X-ray showed a break extending through both orbits, bridge of nose and ending on each side just back of upper last tooth. The right antrum was full of blood.

As the fragment had a tendency to go backward, throwing the front upper teeth far behind the lower front teeth, and sag down so that the fragments were widely separated, the problem was to

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devise a splint to pull these fragments forward and upward. As I failed to find a record of such a fracture, or the description of a splint to hold them, I called in my dentist friend, Dr. J. G. Lutz, who made and applied the splint as described. This splint was kept on for eight weeks. During this time, the right antrum was opened through the nose, and drained of the pus that had accumulated. Recovery was complete except for a widely dilated right pupil, which remains.

Dr. Lutz has very kindly furnished a description in detail of his splint, so that by following his directions, the splint can be made and applied.

Maxillary Transverse Adjustable Fracture Retaining Headgear and Swaged Metal Splint, as devised and used on a practical case by Dr. J. G. Lutz, Los Angeles, Cal. Material for construction of headgear: Secure four pieces of sheet aluminum 1/16th inch thick, 1 inch wide, and approximately 14 inches long; one piece of block aluminum, ½x2x1 inch. Reduce this on both ends and one side to 1/16 inch and with a slight taper to edges to make an offset of ⅜ inch. The block is then drilled, one hole in each end midway of offset surface, and two holes in the offset side, ½ inch apart, or ¼ inch to side of center to receive a 3/16th inch aluminum taper head rivet.

The thicker part of block is then drilled with two adjustment holes ½ inch apart to extend from side to side to receive a No. 8 gauge wire. Also drill two ⅜ inch holes on the face of the block directly over center and to depth of holes previously drilled for No. 8 gauge wire. Tap out holes to receive a ⅜ inch set screw.

Take two blocks of aluminum ½x½x1 inch. Offset ½ inch of one end to ⅛ inch thickness; drill a 3/16 inch hole in upper third for aluminum rivet; drill hole from side to side of thick end to receive No. 8 gauge wire. Open this hole to end of block so as to allow No. 8 gauge wire to be taken in and out without having to push end of wire through hole, simplifying adjustments. Also drill these blocks for set screws as outlined for larger block.

The headgear is then ready to assemble and shape to head. Drill one hole in one end of two aluminum straps and two holes in one end of another to correspond to the two holes previously drilled in frontal adjustment block side and ends. Rivet strap ends to outside of block, having previously countersunk all holes on side in contact with head, so as to make as smooth a surface as possible at rivet heads.

To shape to head, place frontal adjustment block to forehead and hold firmly to place. At same time bend aluminum straps around

sides to follow as near as possible the line of a hat band; then bend the remaining strap up over the head, following the median line to the occipit or lower edge of the side bands. To end of this aluminum strap, rivet a light leather strap, ½x5 inches at its middle. Cut off the side bands about ½ inch short of contact with the middle band. To each of these ends rivet a ½ inch leather strap with buckle attached, which allows for proper adjustment. Conform and adjust to place; then take the fourth aluminum strap, bend and shape over head from side to side, drill and rivet to center and side straps about midway of ends.

Select a thin piece of good kid leather and cover all aluminum straps, having the soft or unfinished side out.

Now place side adjustable blocks to place by drilling a hole at upper one-third of side strap, ½ inch posteriorly to ends of cross-strap and rivet to place.

Secure two 1½ inch or smaller adjustable jack screws. Cut off screw ends and solder a piece of No. 10 gauge German silver wire to screw ends. Make a complete bend, leaving the ends slightly separated. Flatten and shape end and drill small hole for rivet. Likewise, drill rivet hole in side band midway between frontal adjustable and side adjustable blocks and rivet to place. Allow the side blocks and jack screws to work freely after being riveted to headgear.

Construction of gold plate attachments: Modeling compound impression, metal die. Swage a piece of No. 30 gauge, 22-k. silver alloy gold plate, trim to cover teeth labially, lingually, buccally and occlusially, depending upon the length of teeth as to proper height. Reinforce entire rim with narrow strip of plate gold and flow solder over surfaces to secure proper rigidity; also reinforce with stiff No. 11 gauge wire, extending from heel of plate on either side and extending slightly forward and upward across the palate.

Make two tubes of gold plate, ¾ inch long, with diameter to receive No. 8 gauge wire. Solder same to posterior buccal sides of plate. To underside of plate extending from the incisive edges of the central anterially ⅝ inch, or just to the outside of the lip line, solder a piece of No. 30 gauge gold plate, folded once and burnished over a piece of No. 16 gauge wire at its middle. Remove the wire and draw solder between the two surface to stiffen same, which must be made rigid. The folded end to receive the adjusting wire is 1⅛ inches long and cut to taper to the open ends to be soldered under the incisors, to ⅝ inch. Notch the edge at two places from where the No. 16 gauge wire was removed large enough to receive the end of No. 8 gauge wire. Insert No. 16 gauge wire to see that holes are open for pin. Take two pieces of No. 8 gauge German silver wire about 7½ inches long, and drill one end in each wire to receive No. 16 gauge wire pin.

Ready for adjustments: Place headgear and plate in place, bring broken parts to opposition and hold. Shape the two wires drilled to connect to anterior attachment of plate, around sides of nose and up into adjusting holes in frontal adjusting block, by repeated fittings.

Take two more pieces of No. 8 gauge German silver wire about 10 inches long. Insert ends in tubes on buccal sides of plates, bend and shape to curvature of lips at corners of mouth and back over surfaces of cheeks to side adjusting blocks. Remove and solder these ends in the tubes. Place and adjust again with parts in apposition and lock the adjusting or set screws. Use soft ligature wire to connect ends of jack screws to side wires and complete adjusting with jack screws, which will exert the proper tension as required.

In this particular case it was necessary to pin the appliance to place on the teeth, due to the fact that the teeth were very short, several missing, badly decayed and roots abscessed. The pinning was done with splint in place, and starting with a No. 1 round burr, enlarging to a No. 2 or No. 3, and straightening hole with a tapered enamel burr. Make tapered pins and cement to place (leaving pins long enough to be grasped by pliers for removal), one in hole of each central and one in the first bicuspids about one-third up from incisive or occlusal edge, and to depth of approximately 2 m.m.

The appliance was put to place eight days after the occurrence of fracture and was worn for eight weeks with very little discomfort or the slightest kind of an adjustment during the time of its use.

Zinc sterate was used to overcome any skin irritation due to perspiration under the leather pads.

1308 Detwiler Building.

PUNCTURE-IRRIGATION OF THE SPHENOIDAL SINUSES WITH A NEW INSTRUMENT.*

DR. A. LOBELL, New York.

Irrigation of the paranasal sinuses is a recognized and established procedure for diagnostic and therapeutic purposes. As a diagnostic aid, it has no equal in certainty, and rapidity for determining the presence or absence of pus within the sinuses. As a therapeutic method it has a great field of usefulness.

Methods of Irrigation. A sinus can be irrigated by the introduction of a canula into the ostium or by puncturing one of its walls with a needle and forcing the return flow through the ostium (Fig. 1). The former may be designated as the direct method and the latter as the indirect method.

The direct method has been employed in the frontal, maxillary and sphenoidal sinuses. Owing to anatomical variations and technical difficulties the direct method is only successful in a certain percentage of those attempted. This is especially true of the sphenoidal sinuses. The indirect method has almost replaced the direct method in the maxillary sinus. The splendid results obtained in this particular sinus is so well known that it is hardly necessary to dwell on this point.

Comparison Between the Maxillary and Sphenoidal Sinuses. During the embryological development of the sinuses the maxillary appears anteriorly, the sphenoidal posteriorly, while the ethmoid cells are between these two. As a result of this arrangement, in adult life, both sinuses are apt to become reservoirs for pus coming from the anterior and posterior cells, respectively. The ostia are located in an unfavorable position for spontaneous drainage. Stagnation is usually the result.

The maxillary sinus is advantageously located and therefore easily accessible. The sphenoidal sinus has the disadvantage of being located posteriorly and is comparatively inaccessible. This causes delay in diagnosis and treatment until serious complications occur.

The sphenoidal sinus differs from the maxillary sinus in being surrounded by the meninges, the cavernous sinus and its contents, the carotid artery, the hypophysis, the optic nerve and the chiasma. These vital structures are subject to involvement as a result of

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pathological conditions arising in the sphenoidal sinus. Realizing this situation, it becomes our duty to establish methods that will aid us in making earlier diagnosis and in instituting therapeutics in the stage when the pathological condition is still limited to the mucous

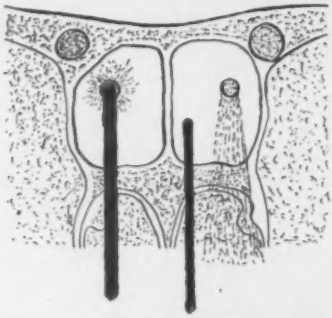

Fig. 1. Diagram illustrating the direct and the indirect methods of irrigating the sinuses. Compare the narrow space for the return flow in the former method with the free exit through the ostium in the latter. In those cases where there is thick pus and a coincident dehiscence in the sinus wall, the undue pressure required would cause unusual symptoms such as Skillern mentions.

Fig. 2. Sagittal section showing a sphenoidal sinus of average size. The anterior wall is thin, the posterior wall has the usual spongy body.

membrane. Therefore, I propose the adoption of the indirect method for the irrigation of the sphenoidal sinuses.

PREMISES UPON WHICH THE SAFETY AND PRACTICABILITY OF THIS METHOD IS BASED.

1. Shaefer and Loeb have studied the dimensions and the capacity of the sphenoidal sinuses. They found that the majority of the

sinuses are of average size and among the remainder large sinuses are more numerous than small ones (Figs. 2, 3, 4).

2. Observations on numerous X-ray plates and operations of the sphenoidal sinuses at the Manhattan Eye, Ear and Throat Hospital corroborate the above findings.

Fig. 3. Section showing a large sphenoidal sinus. Both anterior and posterior walls are thin.

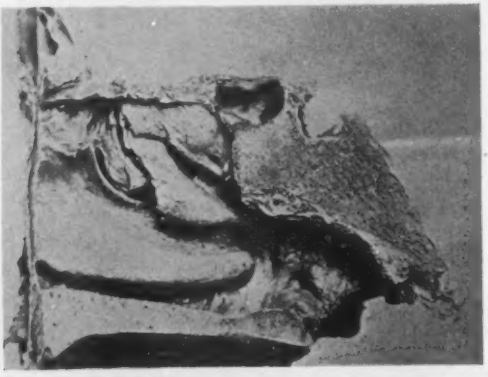

Fig. 4. Section showing a small sphenoidal sinus located abnormally high. Lacking the required measurements, this sinus cannot be irrigated.

3. Small sinuses are infrequently involved because they are located on a level with their ostia, their drainage is ideal. The reverse is true of the large sinuses (Figs. 3, 4, 13).

4. During the development of the sinuses the cavity increases at the expense of the walls, the larger the sinus the thinner the walls, and vice versa (Figs. 3, 4, 13).

5. The anterior wall of the sinus is divided into two parts, the ethmoidal and the nasal. The latter is close to the septum and is exposed in the nasal fossa. This makes it accessible without removing the middle turbinate. These facts make the introduction of a needle into the sphenoidal sinus a practical and safe procedure.

Minimum Requirements. Before attempting this method on the living, I had the opportunity to study this subject on a certain num-

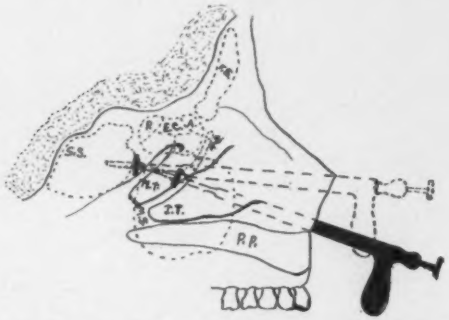

Fig. 5. Diagram illustrating the technic of irrigating the sphenoidal sinus by the indirect method with a special instrument. The oblique direction of the needle is the primary step. Then the needle must be raised to a horizontal line. In this position the wall of the sinus may be safely penetrated. F. S.—Frontal Sinus. S. S.—Sphenoidal Sinus. M. S.—Maxillary Sinus. E. C.—Ethmoidal Cells. A. P.—Anterior-posterior. M. T.—Middle Turbinate. I. T.—Inferior Turbinate. H. S.—Hiatus Semilunaris. P. P. Processus Palatinus.

ber of cadavers. The conclusions are as follows: A sphenoidal sinus is suitable for puncture if the lateral, anteroposterior and vertical diameters measure at least 8, 12 and 16 m.m., respectively. Measurements below these are contra-indications for puncture (Figs. 4, 13). The safest place to puncture the anterior wall is at least 10 to 12 m.m. below the level of the roof of the sphenoid. Another landmark would be at the junction of the middle and lower third of the anterior wall and as close to the septum as possible (Figs. 5, 10). On account of the restrictions, it was found that the introduction of an ordinary needle would be inadvisable. One could not depend on the skill of the operator to control the progress of the needle within the sinus (Fig. 12). It was of utmost importance to devise a special needle for this purpose.

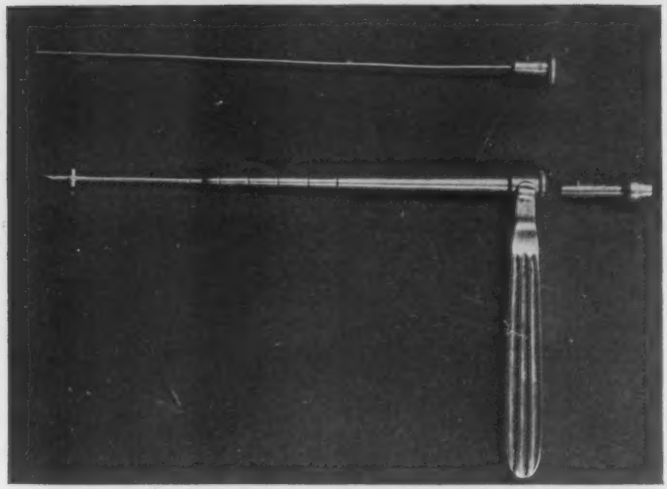

Fig. 6. The author's sphenoidal sinus needle. (a) Needle, shaft and handle. (b) Obturator. (c) Irrigating piece. (d) Automatic stop. (e) Scale in centimeters. The first notch represents 5 c.m., the rest 1 c.m. each. The needle is manufactured by Wregg, Inc., New York City.

Fig. 7. Coronal section illustrating on the right side a marked deviation of the septum that would require correction. The left middle turbinate would have to be pushed aside or infracted outwardly to permit free passage of the needle.

THE NEW INSTRUMENT.

It consists of a needle reinforced on a shaft and joined to an appropriate handle at right angles (Fig. 6). The gage is 16 B.S. The length is 17 c.m. It is notched in such a manner that the first notch from the distal end measures 5 c.m. and each succeeding notch measures 1 c.m. A vertical pin which acts as an automatic stop is

Fig. 8. Roentgenogram. Antero-posterior view showing the roof and floor of sphenoid, thickness of septum, size of olfactory fissure.

located 8 m.m. away from the distal end. It also has an obturator and an irrigating piece.

It is readily seen that the projection within the sinus is less than 8 m.m. because the wall prevents part of the needle from entering. The extent of shortening will therefore depend upon the thickness of the wall. When the needle enters the sinus its progress is not only arrested in an anteroposterior direction, but in addition to this,

its motion becomes limited vertically and laterally. In the hands of the experienced, this device makes the puncture a safe and simple procedure (Figs. 5, 9, 12).

Preparation of the Patient.

1. A rhinoscopic examination may reveal marked deviations of the septum, polypi or other obstructions. These have to be corrected because the most important landmark, the middle turbinate, must be in view (Fig. 7).

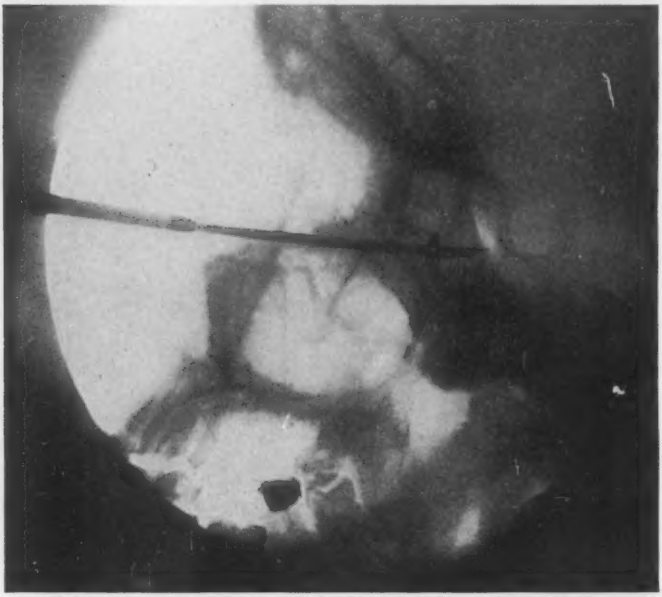

Fig. 9. Rentgenogram. Lateral view. The vertical and the anteroposterior dimensions of the sinus can be studied. Note the location of the sella. The needle is within the sinus; the needle is automatically arrested at the anterior wall. Observe the horizontal direction of the needle.

2. Roentgenographic study: For this purpose we need one anteroposterior, two lateral and one vertical view.

The anteroposterior view shows the vertical extent of the sinus by the lines corresponding to the roof and the floor of the sphenoid. The size of the olefactory fissure should be noted in order to obtain an idea as to how much of the anterior wall is exposed in the nasal fossa (Fig. 8). The lateral view shows the vertical and the antero-

posterior diameters. The thickness of the anterior wall may be seen. The size and the position of the sella should be noted. A stereoscopic examination may reveal the presence of asymmetry, abnormal septa, the thickness of the walls, and misplaced ethmoidal cells (Fig. 9). The vertical view shows the lateral and anteroposterior diameter, the presence of asymmetry and possibly the thickness of the anterior wall Fig. 10). It should be borne in mind that in this view the size of the sinus is magnified to the extent of 1-5. Should there be difficulty in ascertaining the anterior limit of the sinus, a

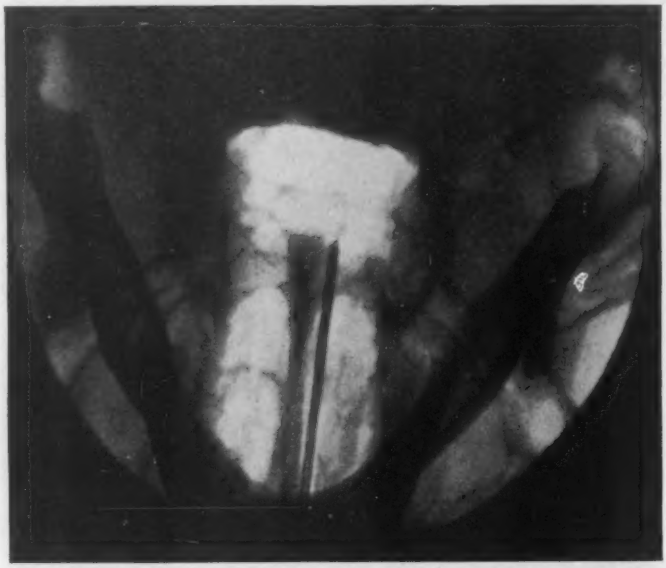

Fig. 10. Roentgenogram: Vertical view showing the lateral and anteroposterior dimensions of the sinuses, magnified 1-5. Note the needle hugging the septum and entering the sinus.

lateral view can be taken with a probe placed against the anterior wall (Fig. 11). Dr. John E. MacKenty has employed this method for exact location of the anterior wall and found it a very valuable guide in operations on the sphenoidal sinuses.

3. *Anesthesia.* In order to anesthetize and to shrink the mucus membrane thoroughly, equal parts of 20 per cent cocain and adrenalin is applied between the middle turbinate and the septum as far back as the sphenoid. Within 5 to 10 minutes, 20 per cent cocain

is applied to the anterior wall of the sphenoid and the region of the sphenopalatine ganglion. The nasal fossa should be thoroughly cleansed when the irrigation is performed for a diagnostic purpose.

Technique. 1. The head of the patient should be placed in an erect posture and it should be properly supported.

2. The obturator of the needle should be lubricated and replaced.

3. With the instrument in the right hand and the thumb resting on the obturator, the needle is introduced into the nose between the

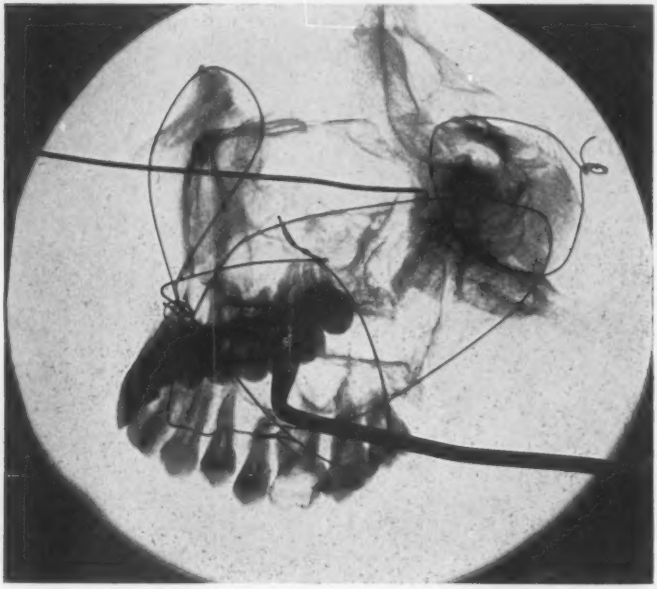

Fig. 11. Roentgenogram showing a probe against the anterior wall of the sphenoid.

septum and the middle turbinate, hugging the septum until it reaches the anterior wall of the sphenoid (Fig. 10). Should the middle turbinate impinge on the septum tightly, it will be necessary to push it or infract it laterally with a long speculum or other suitable instrument (Fig. 7). If the needle is passed along a line extending from the anterior nasal spine and crossing the middle turbinate at its posterior third, it will reach the anterior wall of the sphenoid approximately at the junction of its middle and lower third (Fig. 5).

The distance from the anterior nasal spine to the sphenoid is 7-7½ c.m. It is a good plan to read the needle scale before and after puncturing the wall. At this point the needle is anchored gently, and then the handle is raised to such a degree that the needle lies on a horizontal plane parallel to the roof of the sphenoid (Fig. 9).

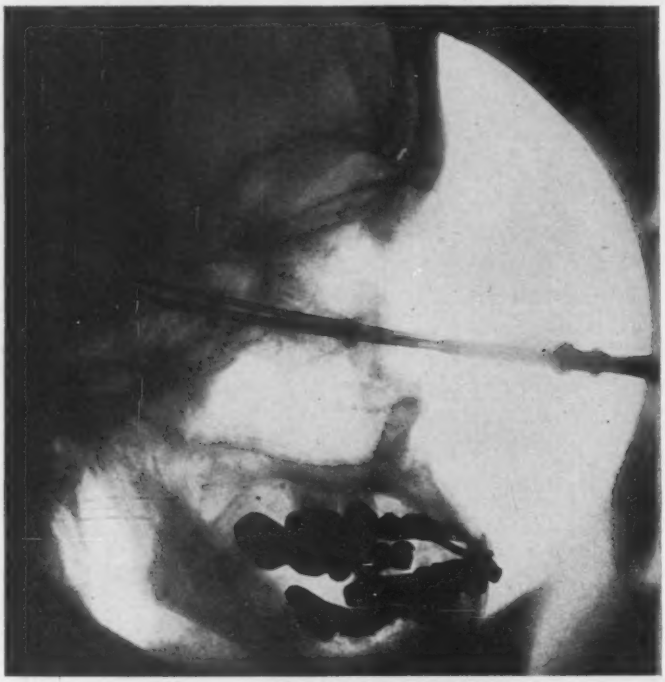

Fig. 12. Roentgenogram illustrating the introduction of an ordinary needle in one sinus and the needle with the automatic stop in the other sinus. Observe the inability to control the former. It went several millimeters beyond the latter without the knowledge of the operator.

Before proceeding to the next step it is necessary to make sure that the head of the patient is in erect posture.

4. At first only slight pressure should be applied to penetrate the wall because the majority of the walls are thin. After breaking in, the needle is permitted to continue within the sinus until it stops automatically (Fig. 9). Occasionally that part of the wall close

to the septum is rather thick and offers resistance. It is then permissible to shift the needle and handle a few m.m. externally.

5. The instrument is transferred to the left hand, the obturator is withdrawn and the irrigating canula to which a rubber tubing is

Fig. 13. Section showing a very small sinus. This sinus has not the required measurements and therefore irrigation is contra-indicated.

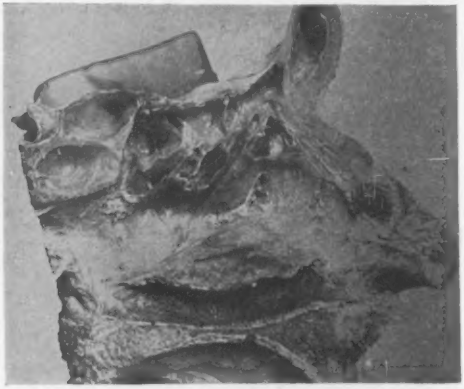

Fig. 14. Section showing two large misplaced ethmoidal cells. There would be no danger if the needle would enter one of these sinuses, because they have the required measurements.

attached is inserted and locked. Dr. Frederick M. Law suggested that in order to ascertain the location of the posterior wall, a fine flexible bougie can be introduced through the needle and X-rayed.

6. A sterile solution is injected by means of a piston syringe. As soon as the operator is convinced that the irrigation can be dis-

continued, the sinus is emptied by forcing through slowly a small volume of air. The instrument is withdrawn and the return flow is examined as usual.

I have performed twenty-two successful punctures out of twenty-four attempted, approximately 90 per cent. In one of the unsuccessful cases an ethmoidal cell blocked the entrance to the sphenoid. In the second one, it was due to a very thick anterior wall.

In all instances there were no complications or sequela. The middle turbinate had to be pushed laterally in two cases, and in one case it had to be infracted. Since this article is only an experimental report, I do not include the clinical histories of these patients.

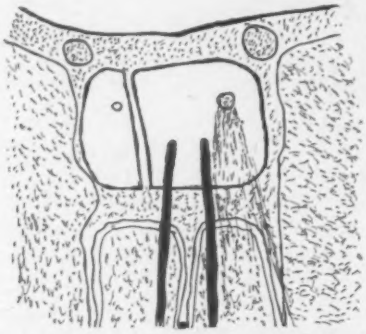

Fig. 15. Diagram to illustrate an a symmetrical sphenoidal sinus. The left sinus is large, extending over the right side, consequently irrigation of this sinus can be accomplished from the right and left nasal fossa by the indirect method.

SUMMARY.

a. The indisputable evidence of the practical value of the indirect method in the maxillary sinuses has paved the way for its adoption in the sphenoidal sinuses.

b. The safety of this method is based upon morphologic, pathologic, and Roentgenographic findings and on experimental demonstrations.

c. This method has the following points in its favor:

1. It is accurate, safe and rapid.

2. In the limited number of cases reported, it was successful in 90 per cent.

3. Very little pressure is required for the injection of the fluid because the return flow has a free exit.

4. It is the method of choice in cases where the ostia are verv small or the pus very thick and inspissated.

5. It is the method of necessity where the ostia are inaccessible; also where there are dehiscences in the sinus walls. In the latter instances excessive pressure would cause untoward symptoms. Skillern mentions unpleasant symptoms that occurred in many cases.

d. The essential points in the technique may be enumerated as follows:

1. The operator must possess a practical knowledge of the surgical anatomy of the sphenoidal sinuses.

2. The correction of such nasal abnormalities that would interfere with the orientation of the necessary landmarks.

3. A Roentgenographic study of the dimensions of the sinuses.

4. Complete anesthesia and shrinkage and a thorough cleansing of the nasal fossa.

5. The employment of a needle with an automatic control.

6. Passing the needle properly and finding the safest point for its entrance through the sphenoidal wall.

7. Raising the handle and shaft to a horizontal line.

8. Placing the head of the patient in erect posture.

9. Careful penetration of the wall, remembering the existence of variations in the thickness of the walls.

10. The use of little pressure and a small volume of air.

I desire to express my gratitude to Dr. John E. MacKenty, Dr. Arthur S. Wilson and Dr. Frederick M. Law for the privilege of using their departments and for valuable suggestions.

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1420 Grand Concourse.

ABERRANT PULSATING VESSELS IN THE PHARYNGEAL WALL.

Dr. Homer Dupuy, New Orleans.

Pulsating aberrant vessels coursing along the pharyngeal wall have more than a simple academic interest. Incising a retropharyngeal abscess, adenoidectomy, and other surgical procedures in that region, when these arterial variations are present, may end in fatal bleeding. An almost tragic experience in my own practice has inspired this contribution.

Case 1: White, female, age 18 years, referred to the Charity Hospital Clinic. She presented a marked left retropharyngeal infiltration. The attending physician had several days before made an incision with no relief to the dysphagia. The swelling did not apparently pulsate. There was a plain indication for surgical relief. I made an incision. Some pus and blood exuded. In a few seconds there was a profuse gush of blood. Mouthsful of blood followed in quick succession. In less than a minute I had applied pressure which promptly stopped the alarming bleeding. The slightest release in pressure brought forth more blood. The patient fainted. We placed her in a prone position on the floor. At the call for help, Dr. P. Graffagnino, House Surgeon, responded. The precarious condition of the patient, and the impossibility of releasing the pressure, without precipitating immediate disaster, forced our decision to ligate the external carotid on the spot. Notwithstanding the unfavorable environment and the position of the patient on the floor, Dr. Garffagnino, by the most skillful and rapid surgery, performed the ligation of the external carotid. The results were gratifying. On removal of the pressure no more bleeding followed. Despite the enormous loss of blood the patient quickly recuperated. The pharynx soon returned to normal. There was no visible pulsating vessel on the pharyngeal wall. Unquestionably this subject must have had a large aberrant vessel within the wall of the pharynx on the left side. It may have been the ascending pharyngeal. Luck favored the physician who first incised. I caught the patient when the element of time had caused the pus collection to gradually erode the externa of the vessel. The evacuation of the pus lessening the counter-pressure a break occurred in the vessel wall, with a near tragedy as the sequel.

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I have observed these vascular anomalies in seven subjects. Five were in adults, one in a child, age 10 years, the other a girl, age 18 years (Case 1). In four subjects, two aberrant vessels presented, one on each side of the lateral pharyngeal walls. In three the vessel was only on one side, the right side. As this is the usual anatomical position of these arterial variations, we offer the following explanations:

1. The anomaly may not be readily observed in a routine throat examination as the vessel, in my experience, is in the lateral pharynx, screened by the posterior tonsillar pillar. Enlarged tonsils in children would especially conceal the danger. Thus it is that textbooks,

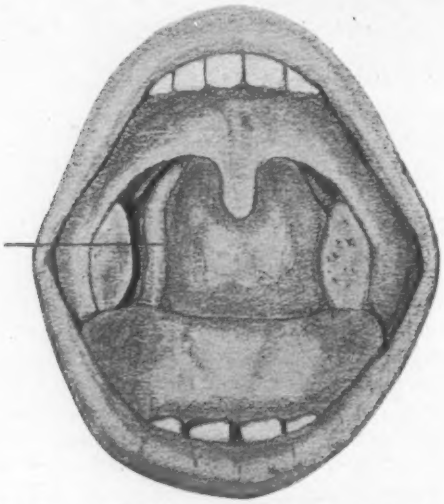

Fig. 1. Drawn by Dr. Jules Dupuy. Shows aberrant vessel in right lateral pharyngeal region. Subject was a white female, age 60 years.

excepting Kyle's 1914 edition, ignore the existence of such anatomical variations. This condition, however, is not unknown, as is attested by a few case-reports in the current literature. Schaffer[1] in a notable contribution gives his anatomical researches on this subject.

2. There were no symptoms referable to these aberrant vessels; the patients knew nothing of their presence. And yet the pulsations in a woman, age 70 years (Fig. 1), were so marked to vision and touch, that we wondered why she did not subjectively feel these pulsations. Unless we are endowed with the traditional eagle

eye of the ideal surgeon, these dangerously situated vessels may escape the usual perfunctory throat examination.

Anatomical considerations: Theoretically, we are supported in our belief, that the internal carotid and ascending pharyngeal arteries sometimes take these anomalous courses. Schaffer, on his dissection evidence believes that the anomalous position of the internal carotid is the chief factor in many cases of large, pulsating vessels in the pharyngeal wall. My case[1] is certainly the exception, as the ligation of the external carotid completely controlled the hemorrhage. My experience opens an avenue for further anatomic investigation. For it

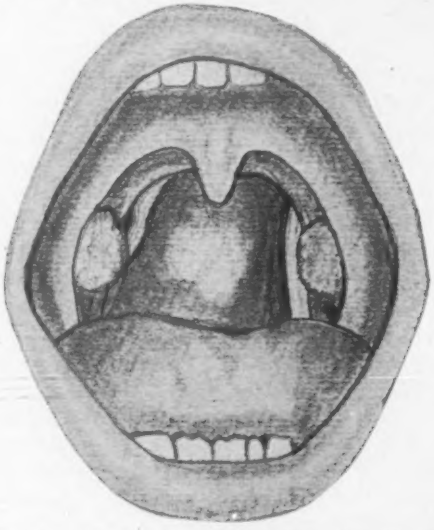

Fig. 2. Drawn by Dr. Jules Dupuy. Shows aberrant vessels in both lateral pharyngeal regions. Subject was a white female, age 65 years.

is of supreme importance when confronted by such an impending disaster whether to ligate the internal or the external carotid. It is well to remember that the internal carotid is really a terminal branch of the common carotid. Briefly, under normal conditions the external carotid gives off the intrinsic blood supply of both the tonsillar and pharyngeal regions. The ascending pharyngeal is normally the closest large vessel to the lateral pharyngeal wall. Theoretically, it should the more frequently follow an anomalous course in the pharynx. Such a plausible view would justify the practice of ligating the external carotid when facing a grave hemorrhagic

situation in surgery of the pharynx. Normally the internal carotid gives off no branches. As the ligation of the internal seriously interferes with the cerebral circulation, even temporarily, the indication in pharyngeal hemmorhagic problems can be usually met by ligating the external carotid.

When discussing such life or death problems it is of importance to remember that after ligation of the common carotid, 20 to 25 per cent of subjects present some intracranial complication from the impaired blood supply. Placing a ligature around the common carotid is technically easier, but it may lead to an unfavorable sequence. In pharyngeal vascular anomalies it is a safer practice to ligate the external carotid, as it offers a larger prospect of cutting off the blood supply vessel.

Conclusions: *1* Pharyngeal vascular anomalies are not generally known and appreciated by those doing surgery in that region. We cannot overstress the clinical importance of this knowledge.

2 There is need for further anatomical studies relating to these vascular variations in the pharyngeal wall. Is the aberrant vessel the internal carotid, or is it a branch of the external carotid? A life problem will, sometimes, depend on the accuracy of this knowledge, and on its application.

3 The author's personal experience (Case 1) rather favors the viewpoint of regarding these arterial anomalies in the pharynx as being supplied by the external carotid artery. Therefore its ligation will usually control fulminating hemorrhage in surgery of the pharynx.

4 All of us who operate in the faucial and pharyngeal field should, in our routine throat examinations, be on the lookout for these dangerously situated vessels. It would serve the purpose of safeguarding our patient, and by increasing the number of observed pharyngeal arterial variations, will lead to a better study and appreciation of these conditions.

5 Operators in this domain of surgery should master the technic of ligating the external carotid in the superior carotid triangle. We really cannot escape this responsibility.

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POST-TONSILLECTOMY HEMORRHAGE DUE TO ATROPIN.

Dr. Casper W. Jennings, Greensboro, N. C.

In our experience, the following case is the first tonsillectomy hemorrhage that we feel could be laid at the door of atropin sulphate; moreover, we have not seen any reports of similar cases in the literature. So, we feel that the following case should be of sufficient interest to justify reporting it.

Report of Case: J. C. H., male, age 45 years, dentist, came under our care Sept. 9, 1925, complaining of mild arthritis in knee joints and frequent attacks of tonsillitis, having just recovered from an acute attack recently. Examination of the tonsils showed no acute inflammation, but considerable evidence of chronic disease.

On Sept. 12, coagulation time was 3 minutes, Wassermann test was negative, and there was no pathology shown in urinalysis. Heart was normal, with pulse rate of 80. Blood pressure was 122 systolic and 85 diastolic. Physical examination was otherwise negative.

On Sept. 16, local tonsillectomy was done at St. Joseph's Hospital, ½ per cent novocain with adrenalin being used. Previous to the operation the patient was given two injections, consisting of morphin ⅛ gr., with 50 per cent magnesium sulphate and 2½ per cent novocain. These were given one-half hour apart, the last being administered one hour before the operation. Tonsils were removed without difficulty. There was a slight ooze from about the middle of the outer wall of each fossa, which was controlled with hemostat and sutured.

Patient spent an uneventful day and when seen at 7 p. m., both fossae were inspected and found to be perfectly dry. A hypodermic of ⅙ gr. morphin and 1/150 gr. atropin was ordered for 8 p. m., in order that the patient might rest well during the night. At a quarter to nine I was called over the telephone and told that he was spitting blood right along and had vomited blood twice. This bleeding had started at about 8:30, or thirty minutes after the injection containing the morphin and atropin.

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On arrival, I found patient with a flushed face and much excited. The pupils were partly dilated, the patient said that his vision was blurred and that he felt very hot. His pulse rate was 140.

Examination of the throat showed the right tonsillar fossa filled with blood and much blood in the retropharynx. Several sponges were inserted in the fossa, but the first three had to be removed on account of the patient vomiting.

After the stomach was emptied, a sponge saturated with thromboplastin was held in the right tonsillar fossa for twenty minutes. There was no bleeding around the sponge. While this was in place sewing material and instruments were gotten ready. However, when this sponge was removed the fossa was dry and gave no hint as to the exact points that had been bleeding. The throat was re-examined thirty minutes later and there was no sign of oozing.

It was then after 11 o'clock and the atropin had worn off and the pulse slowed down and there was no flushing of the face. In the meanwhile, the patient told us that he was sensitive to atropin and that the last dose he had had, two years previously, had made him very sick. Although he was told that we were going to give him something for his pain, he failed to inform us of this beforehand. He had no recurrence of the hemorrhage and went on to uneventful recovery.

We feel that this hemorrhage was entirely due to the whipping up of the circulation by the atropin in a patient who was very susceptible to its effects. We shudder to think what might have happened had we given him the usual preliminary hypodermic of morphin and atropin as we did to all adult patients up until about six weeks previous to the time of this operation.

Clinic Hospital Building.

IS A DIFFERENTIAL DIAGNOSIS BETWEEN MIDDLE AND INTERNAL EAR DEAFNESS POSSIBLE?

Dr. A. G. Pohlman, St. Louis, and
Dr. F. W. Kranz, Geneva, Ill.

The helpful treatment of a given disability is dependent on a fundamental understanding of its causes and in the physical and chemical adaptation of the individual to its results. A definite knowledge of the causes of cataract might, for example, lead to an earlier recognition and perhaps to an ameliorative or preventative treatment. A cataract once formed, quite irrespective of its causation, presents an opaque barrier in the optical system of the eye. An adaptation of the individual with a cataract would therefore logically follow one of three possibilities: first, the discovery of a method to overcome the opacity of the lens; second, the operative removal of the barrier with proper correction of the necessarily altered refraction; and third, the activation of the retina through some other route than the normal pathway through the pupil. The failure to find a method of clearing the lens leaves but two possibilities, and inasmuch as a functional retina is dependent on a reasonably light-proof housing, removal of the lens is indicated and is successfully practiced.

Apart from the subjective responses through the eye to well established and scientifically accurate optical conditions, the observer may examine the refractive media of the patient's eye direct by means of the ophthalmoscope. This is necessarily impossible in the case of the sense of hearing because the observer may neither see his way nor hear his way through the patient's acoustical system.

The invention of the electrical audiometer has given us a definite measuring stick for acuity of hearing. The audiometric method is, however, not directly comparable with the Snellen test-card. The Snellen type is based upon the mathematical relation of the size of the letter to the angle intercepted by the object observed. Audiometric methods are necessarily dependent entirely on subjective registration and therefore may only be standardized on the basis of collective data of individual tests on normal hearing people.

Again, the eye is a slowly operating type of confusion organ and many combinations of colors may produce the same results. The

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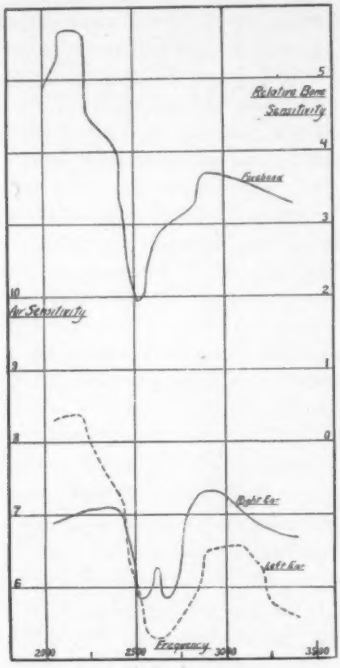
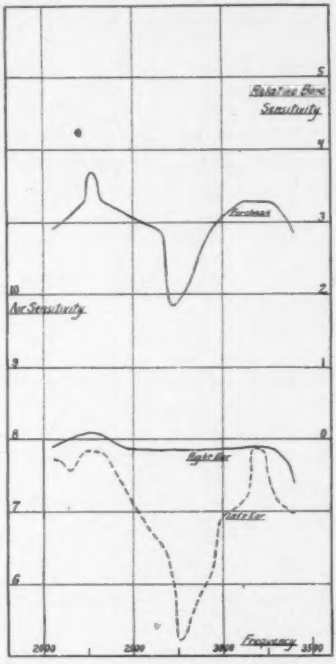
ear, on the contrary, is a rapidly operating organ of analysis and combinations of tones are resolved into the distinct components. One would not anticipate much advance in physiological optics if all researches were confined to the histology of the retina. Indeed the advances in physiological optics have been made in spite of a failure in the solution of the riddle of the retina. Similarly one would not expect great advances in physiological acoustics if the investigations were limited to the microscopic structure of the end



organ. In fact, despite the extensive researches on the end organ of Corti, advances in the treatment of deafness have been conspicuously inconspicuous.

The greater part of the failure in the investigation of the sound transmitting apparatus has been due to the limited number of tools available for such research. The electrical audiometer has given us a quantitative method of determining minimum audition, and within the past five years has yielded much information.

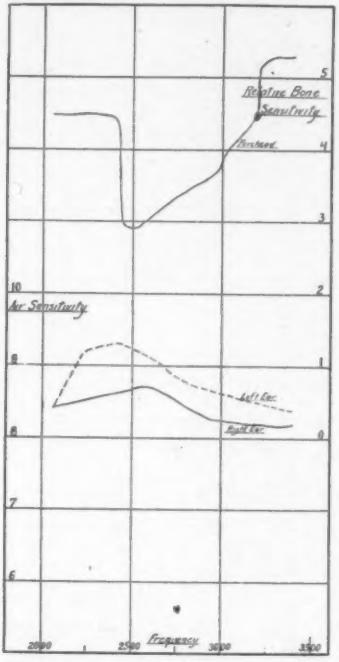
The otologists have built up a number of tests based on the use of the tuning fork; the Weber-Wheatstone, the Rinne, the Schwabach, the Gelle, the Lucae and others. Such tests, when associated with the spoken voice and the whisper, have been employed in an attempt to locate the given impairment in hearing. Two types of deafness have been established, the conduction (middle ear) deafness and the perception (internal ear or nerve) deafness. Insofar as treatment is concerned, no great advance has been made since



the invention of the Politzer bag. Insofar as prognosis is concerned, it seems to make but little difference whether the diagnosis is perception or conduction deafness. Chronic progressive deafness, therefore, presents not only a most pitiful condition, but is an open challenge for an intensive investigation of the problem—a problem directly connected not only with the happiness of many unfortunate individuals, but also one of tremendous economic importance.

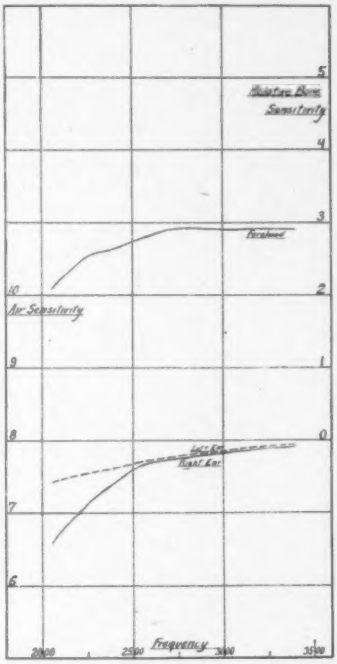
The writers have already published information on the development of the bone activating telephone receiver[1] and also on the binaural minimum acuity for air transmitted sound.[2] We have also in press in the Annals of Otology, a series of articles on quantitative studies in bone and air acuity in individuals with normal hearing. These tests were made on the continuous scale audiometer devised by Kranz[3] at the Riverbank Laboratories.

We arrived at the conclusion that both bone and air transmitted sound normally enter the labyrinth through the oval window. This



bears out the idea proposed by one of us[4] that the end organ is remarkably well insulated against all vibrations except those passing through the stapedial footplate. The testing of air and bone transmission therefore resolves itself into a testing of the conduction system and does not in itself necessarily differentiate between a conduction and a perception deafness. The differential diagnosis between the two types of deafness may be empirically convenient and even time honored, but it is not necessarily correct.

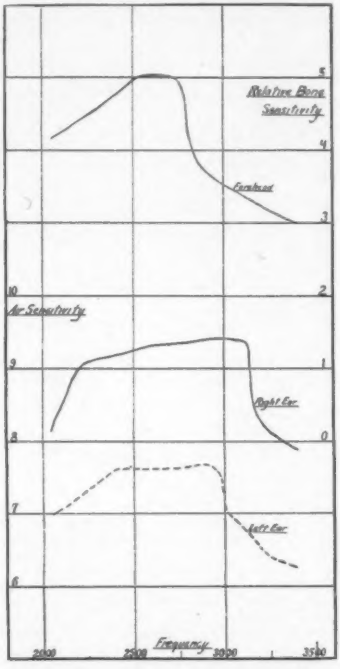
This paper presents additional evidence concerning the ranges of decreased acuity similar to the tone dead areas of Bezold. A range of decreased air acuity if often found in older or matured individuals in the neighborhood of 2000 to 3000 p.p.s. It appears to occur somewhat more frequently on the left side than on the right. If one should find a range of decreased acuity, covering perhaps two or three semitones in the scale, which required a relatively large sound intensity for a threshold stimulus when compared with ad-



jacent ranges (say a factor of 100 to 1000), one would be tempted almost offhand to class it as a perception deafness. Further, if a deficient range of this sort maintained itself for a number of years, and we have found it five years in one individual and four years in another, one would feel that one's suspicions were confirmed. One would, according to accepted ideas, picture a lesion in some particular part of the end organ and one would anticipate structural changes in this particular location in the organ of Corti.

Can this interpretation be tested? We present in this paper, evidence obtained on several individuals, which we believe sheds some light on the validity of this hypothesis.

The continuous auditory field of frequency is shown for Case 1 in Fig. 1. The air acuity curves are plotted in absolute units, based on the necessary intensities measured in ergs per square centimeter per second. The scale at the left of the chart shows the logarithms of the reciprocal of this necessary intensity, which gives



what is probably the best scale yet devised for sensitivity. The bone acuity curve is shown in relative intensity units which are the same for all the individuals reported. The charts are made in such a manner that the lower the curve, the poorer the acuity. The observations on bone sensitivity were all made with the bone activating telephone on the forehead.

Fig. 1 shows a fairly well developed bilateral deficiency for air transmitted sound around 2500 to 2700 p. p. s. Partial recovery

takes place at 2900. The bone acuity curve appears to follow in a general way those of the air acuity.

This case might have been interpreted as one of a local lesion of the cochlea which interfered with sound reception. Indeed, some investigators might even have gone so far as to indicate diagrammatically a certain area on the basilar membrane which should be the seat of this small sharp deficiency. They would have regarded the possessor as of considerable histological importance.

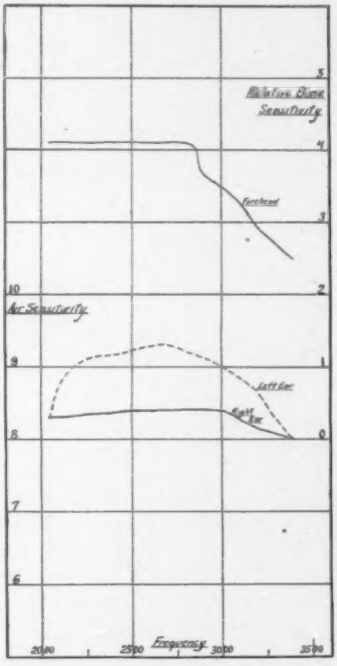
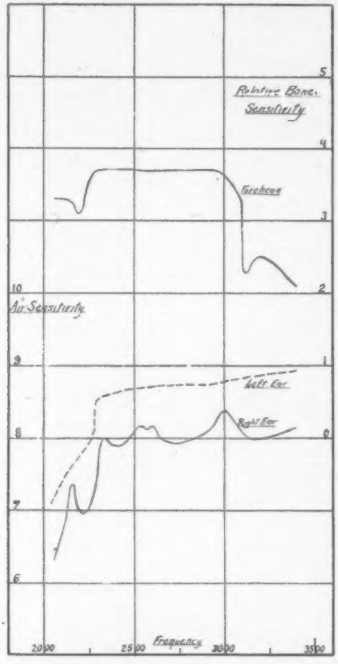


Fig. 2 shows the bone and air acuity of Case 2. The right ear is practically normal for air transmitted sound. The left ear presents a conspicuous drop off in acuity with a minimum of 2750 p. p. s., with partial recovery at higher frequencies. The bone acuity curve follows in a general way that for the air acuity of the left or poorer ear.

Now, while Case 1 seems to fall in with our generalization made elsewhere (i.e., that good bone acuity is more commonly associated

with good air acuity than with poor air acuity), the curve for Case 2 appears to show that the hearing of the bone transmitted sound followed the poorer ear. The actual tests in the subjects were made in the same order as presented in this paper. After considering the results on Cases 1 and 2, it seemed possible that the deficiencies indicated in the bone sensitivities were due to peculiarities in the bone activating telephone receiver. The characteristics of the instrument had not determined, although it is true that the maxi-



mum deficiencies are not at exactly the same frequency for the two subjects.

Case 3 was selected because it was known that this individual had practically normal hearing in both ears for air transmitted sound. This is shown in the air sensitivity curves of Fig. 3. The bone acuity curve, however, shows a well defined range of lowered sensitivity with a maximum deficiency at about 2500 p. p. s. Case 3 seemed to confirm the suspicion of a peculiarity in the response of the bone activating receiver in this particular range.

For further confirmation of this, another individual with normal hearing through the range in question was selected and the curves are shown in Fig. 4. While the air sensitivity curves of Cases 3 and 4 are quite similar, Case 4 shows no tendency toward a deficiency in the bone sensitivity such as found in the three preceding subjects. This led us to test three more individuals and the results are given in Figs. 5, 6 and 7. It is seen that none of these latter cases show any depression in bone sensitivity in the middle part of the range used. The most reasonable explanation is that Cases 1, 2 and 3 had actual deficiencies in their bone sensitivities in the middle part of this range.

All seven cases were retested with a bone activating receiver of entirely different construction and undoubtedly with considerably different characteristics, and the bone acuity curves thus obtained substantiated the results given above. A few weeks later the original tests were repeated, again giving a confirmation of the above results.

DISCUSSION.

Cases 1, 2 and 3 showed similar deficiencies in the bone sensitivity curve, which might reasonably be regarded as due to a common cause. The possibility that the dip in the bone acuity curves might be due to the peculiarities of the bone activating instrument used, was eliminated in two ways: first, four other individuals did not show this dip; and second, the results were confirmed for all the individuals using a different type of receiver.

Case 1 might perhaps have been explained on the basis of a theoretical cochlear lesion. This possibility seems to be eliminated, however, through the findings on Case 3, where the deficiency in bone acuity is similar to that found in Case 1, but in which the air sensitivity is normal for both ears.

Case 2 might be considered as a possible exception to the generalization that the bone acuity usually follows the curve of the air acuity of the better ear rather than of the poorer ear. This explanation is, however, not substantiated by Case 3, in which the poor bone sensitivity was not associated with poor air acuity in either ear.

The possibility therefore suggests itself that Case 2 was really hearing the bone transmitted sound with the ear which was most sensitive for such bone transmitted sounds, and that the right ear of Case 2 and the two ears of Case 3 are in a similar condition of deficient bone acuity. Case 3 is the youngest of these three individuals and there is a possibility that the right ear of Case 2 and the two ears of Case 3 may eventually show the same lowered sensitivity for

air transmitted sounds as is found in Case 1 and in the left of Case 2.

We regard this evidence as substantiating our previous statement that both air and bone transmitted sounds normally enter the cochlea through the stapedial foot-plate. On the basis of this and other experimental work done at the Riverbank Laboratories, we are inclined to doubt whether it is possible, at the present time and with the present methods, to definitely differentiate a perception from a conduction deafness. When our knowledge of the acoustical system has advanced sufficiently, we may be able to establish exactly where an interference is located in the sound transmission apparatus and a technique may then be developed to adapt the individual to his defect. It may also come to pass that many cases which have been classed as perception deafness or nerve lesion may be benefitted through operative procedure.

It may be pointed out that the present paper serves as an example of the researches made possible by the use of the continuous scale audiometer, yielding information which an audiometer of discreet frequency intervals would never have furnished.

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THE DIAGNOSIS OF TUBERCULOUS OTITIS WITH REPORT OF A CASE.

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Tuberculous otitis is important, not only because of its known frequency, which is such that almost every otologist will meet cases of it, but also because, if the process is not checked, it can lead to very serious intracranial complications. It is very desirable, therefore, that not alone otologists, but every physician should be acquainted with the diagnosis and differentiation of tuberculous otitis, and it is with a view of pointing out the chief factors that enter into diagnosis that this paper is presented.

In the presence of any purulent middle ear discharge it is essential to verify whether or not it is of tubercular nature.

Before entering into the general discussion we desire to report the following case in which the tubercle bacillus was isolated from the middle ear secretion.

Case Report: J. R., age 21 years, single, born in United States. Pulmonary tuberculosis with laryngeal involvement three years ago confined this patient to a tuberculosis sanatorium for one year. Two years ago patient upon leaving the hospital as an arrested case, continued the use of sunlight at home for the laryngeal involvement.

For one year patient has felt his hearing was diminished in the left ear. Upon examination the left membrana tympani was colored a marked brownish red, and apparently quite thickened. At no time was there any pain. The hearing for tuning forks C_5 (4096 double vibrations) and C_4 (2048 double vibrations) was diminished; the Rinne was negative and the Schwabach was referred toward the involved side. The hearing for the whispered voice was reduced to 0.5 M. The right ear was apparently normal as far as whispered voice and tuning fork tests could indicate.

A parcentesis was done to relieve what seemed to be a serous otitis; smears made during the first few days from a thin purulent secretion and stained for tubercle bacilli demonstrated these in large numbers. The patient is still under observation.

Tuberculosis otitis may be met with either in the child or in the adult, but is far more frequent in children of early age. In the adult it is more likely to be associated with advanced pulmonary

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tuberculosis. Turner and Fraser[1] state that the incidence of suppurations of the ear which are tuberculous vary according to different authors from 27 to 50 per cent in the two first years of life, but rapidly diminish in the case of older children.

Bezold[2] considers that tuberculous otitis constitutes only 4.4 per cent of all chronic otorrheas. Brieger[3] in 431 autopsies of tuberculous subjects found chronic otorrhea in 22.7 per cent, and of these, 14.3 per cent were tuberculous.

Lautmann[4] states that more reliable evidence is obtained from the operative side. He cites Busch, who in 60 antrotomies, done within two years, found 10 per cent of tuberculosis based on examination of the granulations, that a similar operative percentage was found by Siebenmann, and that Voss in examining the granulations of 300 middle ear operations found 16 to be tubercular, although in many, clinical evidence of tuberculosis was absent. Henrici[5] thinks that one-sixth of all mastoiditis in children are tubercular and that tuberculosis of the mastoid apophysis is specially frequent in children.

It is difficult to obtain any reliable data in regard to the frequency of auricular tuberculosis associated with tuberculosis elsewhere.

Perhaps the best general ideas as to the incidence of tuberculous otitis will be gathered from recently published personal statistics of Leegaard[6] and Nakamura.[7] In 200 personal cases of purulent otitis Leegaard, either histologically or by animal inoculation, demonstrated the tubercle bacillus in the tissues in 20, i.e., 10 per cent. These cases were mostly in children from 3 to 7 years old. In 80 cases of various types of middle ear infections, Nakamura, either by cultures or inoculation tests, demonstrated the tubercle bacillus in 23.75 per cent, and in about half of these there was a coincidence of pulmonary tuberculosis. These figures show that even with the most painstaking efforts there is still a wide divergence in the findings.

Tuberculosis of the ear is usually admitted to be secondary from some other tubercular focus in the body. Lagarraga,[8] Orioli[9] and Henrici[5] and other writers think it may be primary, the infection reaching the middle ear by the vehicle of the blood. Henrici was quite convinced of this mode of infection in a case of tubercular mastoiditis without any evidence of a prior otitis. Generally if the tube and tympanum are found exempt at operation the infection may be presumed to have been through the blood.

According to the views of those who support the possible hematogenous route of tubercular infection of the middle ear, the bacilli first locate in the bone and thence spread to the mucosa of the drum.

But as already stated, most writers agree with Brieger's theory that infection of the middle ear is secondary through the Eustachian tube, and that most frequently the original tubercular focus is pulmonary.

In the diagnosis of tuberculosis of the middle ear clinical symptoms, while suggestive and presumptive, are not conclusive. A number of such clinical symptoms have been reported, some or all of which may be present in a given case. These symptoms include: multiple perforations of the tympanum; miliary tubercles or granulations developed on the tympanum; purulent discharge; absence of pain in the earlier stages; facial paralysis; enlargement of the neighboring glands; stapedial damage; marked loss of hearing; rapid destruction of tympanic membrane; presence of a fibrinous layer over the promontory, a sign which has been emphasized by Herzog[10] and Bezold.

Leegaard found multiple tympanic perforations only once in 25 actual cases.

Facial paralysis has been considered by many as a frequent symptom and occurring in about one-third of all cases, but Leegaard thinks it of value only in advanced cases. He did not find it in any of his 25 proved cases.

If several of the foregoing signs are recognized in a patient who is otherwise manifestly tubercular, there would be strong presumptive evidence of auricular tubercular involvement. Generally, however, cases of auricular tuberculosis present themselves to the otologist in the guise of an ordinary purulent otitis media and some method of distinguishing its nature must be employed. These measures include: *a.* search for the Koch bacillus in the aural discharge; *b.* histological examination of excised granulations or other tissue, including dead bone; *c.* reaction to tuberculin; *d.* the Von Pirquet biological reaction; *e.* animal inoculation; *f.* complement fixation test and cytological examination of pus from the discharging ear if present.

The demonstration of the Koch bacillus in the auricular discharge is the most rapid and certain method of diagnosing auricular tuberculosis; but such a demonstration is not always possible even in cases of undoubted tuberculosis. Miligan[11] showed that in cases of acute onset if examination of the discharge is made at a very early date there is more likelihood of demonstrating the tubercle bacilllus. In the more chronic forms of the disease the almost invariable presence of mixed infection tends to destroy any existing tubercle bacilli or to distort and alter them beyond recognition. Lautmann

states that Eschle was the first in 1883, shortly after the discovery of the Koch bacillus, to demonstrate its presence in the discharge from auricular tuberculous cases.

Generally speaking, from what is known of the pathological anatomy it is easy to understand that, in the simple congested and infiltrated types without destruction of the mucosa, the bacillus might be absent. The fibrinoid type is more favorable for the demonstration of the bacillus; and in the discharge from the myringitic type the bacillus is almost always present. Not only may there be absence of bacilli, but even when they are present the ordinary methods of microscopical demonstration may fail. Wingrave[12] in the careful microscopic examination of the discharge in 100 cases of middle ear suppuration showed that acid-fast and alcohol-fast bacilli are very frequently found and that there is a great possibility of error in differentiating the acid and alcohol-fast tubercle bacilli. But with the more recent improved methods this difficulty has been much obviated. Cocks and Dwyer,[13] in 1915, reported an improved method of isolating and cultivating the tubercle bacillus, based on the use of antiformin, which destroys all common pyogenic organisms except the acid-fast group. The cultivation is in a special medium devised by Petroff[14] in 1914 and the authors quoted stated that from examination of the aural discharges in children they concluded that 15.6 per cent of all purulent otitides were tubercular. They admit, however, that there is always an element of uncertainty. The consensus of opinion is that a veritable demonstration of the tubercle bacillus from microscopical examination of the discharge can only be expected in about one-third of the cases of tuberculosis of the middle ear, and a negative result does not rule out tuberculosis, if the clinical evidence is strong.

For the foregoing reason many otologists prefer to rely upon histological investigation of excised granulations or removed pieces of dead bone. A biopsy permits a rapid and sure method of diagnosis. One of the typical granulations, which cause the clinical suspicion of tuberculosis, should be excised.

A histological examination can be made in fifteen minutes and if positive an otologist in the course of an operation can be guided by such findings. But a negative result does not necessarily rule out tuberculosis, and Lermoyez[15] and Tits[16] insist that several repeated examinations may have to be made. Leegaard considers that histological examination of removed tissue and inoculation are the best methods of diagnosis. Histological examination gave positive re-

sults in 13 out of 18 cases examined, and Leegaard considers it more reliable than the search for the tubercle bacillus in the secretions.

The animal inoculation test is perhaps the most reliable of all methods of diagnosis. But it is objectionable from the point of view that it takes too long, requiring perhaps two months before definite evidence is obtained.

A few words may be said regarding local and general reactions to tuberculin. Leegaard considers that cutireactions are valuable in the young, but less reliable in the adult. Generally, such reactions can only be considered as reactions of the whole organism to tuberculosis. Brieger, however, places importance on the reaction to tuberculin injection in the ear. Most writers consider that owing to the formidable reactions to tuberculin (labyrinthitis, facial paralysis, etc.) that this method should be discarded by otologists.

Lautmann evolved a method akin to the rhinoreaction with tuberculin devised by Dupont and Molinié[17] and similar to the tuberculin ophthalmoreaction. Such an otoreaction to tuberculin was also reported by Tovolggi[18] at the International Congress of Medicine, London, 1913, but it does not appear to have attracted much attention in practice.

The complement fixation test and cytological examination of the discharged pus cannot up to the present time be regarded as of any value in the diagnosis of middle ear tuberculosis.

In conclusion, we would say that early recognition of tuberculosis of the middle ear is important on account of the possible serious intracranial complications which can arise from it independently of tubercular lesions elsewhere. According to Milligan these complications may arise: *a.* by the tubercular invasion of the membranes of the brain directly from the local focus; *b.* by the slow disintegrating process followed in the evolution of middle ear tuberculosis which lays bare the intracranial contents and exposes them to attacks of pyogenic cocci, the virulence of which has been increased by the pabulum which the liquefaction of the tubercle bacillary products afford.

Tubercular meningitis can, of course, arise apart from middle ear tuberculosis by independent infection extending from the primary focus.

Possible extension to the labyrinth must also be borne in mind, though it is less rare apparently than tubercular meningitis.

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REPORT OF CASE OF EGG SHELL IN LARYNX.

DR. J. I. BERLIN, Jersey City, N. J.

Patient, R. Z., female, age 9 years, was referred to me by Dr. M., of Jersey City, complaining of complete loss of voice of four weeks' duration, showing no improvement under local treatment, and showin gno other symptoms than the hoarseness above mentioned.

On questioning the patient on May 19, 1925, the day the patient was sent to me (her voice was of a low, indistinct whispering type) no history of a foreign body could be obtained. Indirect examination was unsuccessful because of the extreme nervousness of the child.

May 21, patient was admitted to the New York Post-Graduate Hospital and preparation made for direct laryngoscopy or bronchoscopy as may be found necessary at the time of operation.

On the following day the writer made an examination by direct laryngoscopy, under local anesthesia, and found a piece of egg shell about the size of a five-cent piece, somewhat triangular in shape, hanging downward in the trachea and firmly attached to the right vocal cord, parallel to the cords. Removal of the egg shell was a simple matter. On the following day the patient's voice was more audible, and complete restoration of the normal voice was accomplished in five days.

9 Gifford Avenue.

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BILATERAL MASTOIDITIS IN TWINS ACCOMPANIED BY DENTITION AND COMPLICATED BY FACIAL PARALYSIS. REPORT OF CASES.*

Dr. J. Manuel Mayer, Brooklyn.

Introductory Remarks: Physicians in the middle ages and until the eighteenth century attributed many ailments to teething. Hippocrates ascribed fever and convulsions to the cutting of teeth. In the middle of the nineteenth century, 7 per cent of deaths reported officially in London under the age of 3 years were attributed to dentition.[1] However, Holt states that many observers at the present time firmly deny that dentition is ever a cause of symptoms in children. Although dentition per se is a physiologic process, yet it must be admitted that particularly in delicate and highly nervous children dentition may produce many reflex symptoms, some even of quite an alarming character.[2]

The history is as follows: Both Martha and Gilda, age 4 months, born a half hour apart, weighed about 6 pounds each at birth. Bottle fed, steadily gaining in weight and in excellent health. About Aug. 5 of this year, Martha's temperature registered 103,° which was constantly rising, reaching a maximum temperature of 106.° At this time the baby was taken with convulsive seizures, both tonic and clonic in character. The temperature was fluctuating and the convulsions recurred from time to time. This condition lasted for a period of four days, when the temperature abruptly dropped and two lower incisors made their appearance.

Afer a lapse of a few days there was a recrudescence of temperature, which likewise subsided after a spontaneous rupture of both drums discharging a serosanguinous material.

Hardly had Martha fully recuperated, when her twin sister, Gilda, went through a similar cycle.

On Aug. 25, I was consulted for the first time. The otologic examination revealed the following findings: The ears of both children were discharging copiously a thick purulent material. On wiping, small irregular perforations centrally located were noticed on the drums. The fluid refilled rapidly, but did not pulsate. The canal walls in both children were slightly corrugated. The lobes of

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the ears were totally free. Neither pre- nor postauricular swelling was preesnt. The children seemed comfortable and the mother claimed that they slept well at night. Re-examination of patients on Sept. 2 showed an appreciable loss in weight in both. They were pale and fretful. Sleep at night was now frequently interrupted and the intake of food a good deal lessened. The discharge was profuse, refilled rapidly and pulsations were marked. The posterior canal wall did not droop. Temperature ranged from 100° to 101.°

On Sept. 11, Martha, the first one to become ill, was finally admitted to the Israel Zion Hospital with a moderate amount of swelling over the right mastoid. The postauricular fold was slightly obliterated and sagging of the superior posterior canal wall was marked.

Temperature at this time was septic in character. A right mastoidectomy was performed. Three days later the left mastoidectomy was performed. At the same time, Gilda, her twin sister, was operated upon on both sides.

Laboratory Findings: Smears from the ears of both children showed streptococcus hemoliticus. Martha: leucocytes, 20,000; polys., 81; lymphocytes, 19; hemoglobin, 75 per cent. Gilda: leucocytes, 15,000; polys., 79; lymphocytes, 21; hemoglobin, 74 per cent. (Sahli.)

Urine Analysis: Trace of albumin, otherwise negative. X-ray was not deemed advisable as it is not considered to be of much value in infants, since the mastoid cells are not fully developed at so early an age.

Operative Findings: Cortex of both children exceedingly pale and perforated. Bone diploic and considerably necrosed. Antrum filled with free pus and granulations. Lateral sinus intact. Dura exposed.

Patients were discharged from the hospital after the second dressings with a flat temperature.

About ten days after the operation, Martha, the first one to be operated upon, shot up a temperature of 106° and over. Both wounds were promptly inspected and found to be discharging slightly and rapidly healing. Small masses of granulations were present over the line of incision. Aural discharge was scanty. There was no opisthotonos present. Both Kernig and Brudzinska were negative. Taches cerabrales was not in evidence, yet the temperature persisted. Careful physical examination by a competent pediatrist

was also negative. Urine analysis examined and found normal. The temperature, which lasted for four days, ranging between 103° and 106° finally dropped as two upper incisors appeared.

The wounds of both children were now totally closed and their health apparently restored, when suddenly, Gilda, the second child, became fretful, her little head tilted towards the left, her left face seemed immobile and the left eye was widely open when she cried. A diagnosis of left facial paralysis was made.

Fearing the possibility of bone caries exerting pressure downwards upon the facial nerve, I reopened the wound and drained it for several days. After an elapse of three weeks, the facial cleared up completely.

COMMENT.

I was prompted in reporting these cases, not because I had something new to offer, but merely to elucidate several points of interest, namely: The simultaneous development of disease in both children, affecting also a third one at home, who barely escaped operation; the acute alarming symptoms, such as convulsions preceding the otologic involvement and the acute postoperative pyrexia, which were unquestionably due to dentition, the latter causing serious reflections as to the possibility of meningeal involvement or septic sinus thrombosis; the uncommon occurrence of facial paralysis three weeks after operation.

BIBLIOGRAPHY.

1. Reference Hand Book of Medical Sciences, Vol. 3, p. 492.
2. Diseases of Infancy in Childhood, Holt, p. 268.

5115 15th Avenue.

AMERICAN BOARD OF OTOLARYNGOLOGY.

In addition to the examination held at Dallas on Apr. 19 and at San Francisco on Apr. 27, another examination will be held at the Otolaryngological Clinic, Royal Victoria Hospital, Montreal, on Tuesday, June 1st.

Information may be secured from the Secretary, Dr. H. W. Loeb, 1402 South Grand boulevard, St. Louis, Mo.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

Regular Meeting, November 25, 1925.

Case of Thyroglossal Fistula. Dr. Lee M. Hurd.

J. M. when admitted to the Clinic complained of a discharge from a fistula in front of the neck, the duration of which he did not know.

Examination showed a fistula through the skin, slightly to the right of the midline of the neck, with an external opening over the thyroid notch and running into a small, firm mass just below the hyoid bone, having a thin watery discharge.

Under general anesthesia, the tract being injected with methylene, blue dye and paraffin, a vertical incision was made surrounding the opening of the fistula, and the fistula and mass over the hyoid were dissected out *en masse*. The prolongation running upward between the geniohyoglossi muscles was traced to its limit and removed. Three stitches of silk closed the lower half of the wound, and the upper half was packed with iodoform gauze.

The lower part of the wound healed by primary union, and the stitches were removed on the fourth day. The upper part of the wound healed in ten days, after slight granulation. The result was a firm scar without any mass beneath or fistulous tract remaining. The patient was discharged from the Clinic after three weeks.

Case of Unilateral Chronic Suppurative Pansinusitis Treated by Radical Operation Under Local Anesthesia. Dr. Lee M. Hurd.

C. M., a young negro man, was admitted to the Clinic complaining of purulent nasal and postpasal discharge, with attacks of pain over the left eye and in the left cheek, following an attack of influenza two years previously.

Examination revealed a straight septum, a hypertrophied middle turbinate, with much pus in the left ethmoids and left frontal sinus. X-ray examination showed all sinuses on the right side clear, and all on the left side very cloudy, the frontal sinus being high and deep and extending outward into the external angular process of the frontal bone.

Initial Operation: Under local anesthesia, the left middle turbinate was removed, and the ethmoids were exenterated. There was much pus from the frontal sinus, the opening into which was enlarged. Foul and thick pus continued to be douched from the left frontal and left antrum.

Radical Operation (Mar. 5, 1925: The patient was given a preliminary medication of morphin sulphate, grain ¼, and hyoscine hydrobromid, gr. 1-100, 45 minutes before operation; this was repeated at the beginning of the operation. Iodin and alcohol skin preparation. One per cent novocain with adrenalin was injected into the orbit to the superior orbital fissure and into the second branch of the fifth nerve in the sphenomaxillary fissure from the side of the cheek. A typical radical frontal and ethmoid exenteration was performed, using the Killian technique The frontal was found to be full of foul pus, and the membrane changed to polypoid and granulation tissue. The sinus extended almost to the hairline near the median line, with several partial septa, with no bony necrosis, and several orbital prolongations of the orbital ethmoid cells extending backward almost to the apex of the orbit. Laterally, the frontal extended into the external angular process of the frontal bone. All diseased membrane was removed, and the remaining ethmoid cells were removed, leaving a large opening into the nose from the frontal. The external wound was sutured.

Post-Operative Course: An orbital hematoma developed that pushed the conjunctiva of the globe into large folds, necessitating an external camthotomy and the suturing of the lid margins together for a week; there was quite marked external strabismus and diplopia. The orbital

hematoma was absorbed, and the external wound healed by primary union.

Radical Antrum Operation (May 7, 1925): Preliminary medication of morphin sulphate, gr. ¼, and hyoscin hydrobromid gr. 1-100, was administered 45 minutes before operation. One per cent novocain with adrenalin was injected into the infraorbital nerve, and pharyngopalatine fassa. A tpical Denker operation was performed. The antrum was full of foul pus and polypoid membrane. A large opening was made into the inferior nasal meatus, and a flap of mucous membrane was turned into the floor of the antrum; mouth wound sutured.

After the usual reaction, the mouth wound healed in ten days, and no leakage occurred.

DISCUSSION.

DR. JOSEPH BRUDER: How does Dr. Hurd account for diplopia, ten months after operation? In this connection, it may be of interest to this Section to hear of an experience in Berlin a year ago, showing that operations in the neighborhood of the orbit and ethmoid region are associated with danger to the eye, resulting in blindness from trauma to the optic nerve. Dr. Halle told me of several cases of blindness having been reported following injections of an anesthetic in the orbital or ethmoid region. He brought the matter up at several ophthalmological and Rhinological societies in Berlin, and no one could account for the cause of the blindness. He thought it might be caused by pressure on the optic nerve, due to hemorrhage and hematoma in the orbit, and instructed his men to make a free incision into the orbit, relieving the pressure of optic nerve if it should happen again. In the next case of blindness, during the operation, the incision in the orbit was made, and in that case, the vision was restored. I happened to be present when his assistant was demonstrating the ethmoid operation step by step, on one of Dr. Halle's patients and suddenly noticed a hematoma developing in the orbit. He at once tested the patient's vision, and found him to be totally blind, with no light reaction in the pupil. I made an ophthalmological examination, which I found to be negative, except for a slight hyperemia of the disc. I discussed the case with Dr. Halle, and told him that in my opinion, the pressure of a hematoma alone could not cause such a sudden and complete obscuration of vision and that the optic nerve was injured by pressure from instruments forceps or knife. The result was, that on the fourth day following the operation there was a complete atrophy of the optic nerve. Dr. Halle opened the orbit of the patient to see whether the nerve could be reached from within the nose and found an opening through the lamina papyracea, evidently caused by the instrument, and through the opening, I was able to grasp the optic nerve with the nasal forceps, thereby proving my contention that the blindness was the result of the injury to the optic nerve and not from pressure from hematoma. I have since tried to grasp the nerve through the opening in the lamina papyracea cadaver, and found that I could easily take hold of the optic nerve with the nasal forceps.

DR. HURD: I agree with Dr. Bruder that these injections are not devoid of danger. It is like a flame of fire when you do get them. I got an enormous hematoma of the orbit. The edema of the conjunctiva was so great that I could not get the lower lid over the terribly swollen eye. It is a wonder that he did not have more, and I got that only from pricking the vein. You can probably get enough novocain to get a temporary blindness, but I don't believe that a straight hematoma would do it, but it is not a plaything at all. You have to keep out of the orbit and scrape between the bone and the periorbit until you get beyond. Indeed, I think that the best way would be to go through the cheek, but you do get as complete anesthesia as with ether. At times when I have been here I have seen cases where Killian's operation was done with stress laid on the fact that you have to have a pad over where the frontal wall was taken out, and have the scalp kept in close contact with the posterior wall or you will have something happen. This man had nothing of the kind, and he has nothing up here—no pressure whatever. That will heal

up tight, without any adhesions. If the patients want it, they can have some paraffin injected, but usually they don't mind—but such injection around the orbit is playing with fire.

Lateral Cervical Fistulae. Dr. Rudolph Kramer.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Pharyngeal Stenosis Treated by Endothermy. Dr. Arthur Palmer.

Mrs. A. H., age 38 years, born in Bohemia, was a patient from the service of Dr. Harmon Smith in the Department of Laryngology at Cornell Clinic, referred from the Department of Dermatology, where she was being treated for syphilis. When examined on Apr. 24, 1924, her chief complaints were inability to breathe through the nose and difficulty in swallowing. She gave a history of syphilitic symptoms when 17 years of age, and some treatment in 1910. She had a healthy child in 1911. In 1917 she had more treatment, and after a while the Wassermann reaction was said to be negative. She had difficulty in swallowing during this period. On Mar. 25, 1924, her Wassermann reaction was four plus. Examination of the throat showed an almost complete stenosis between the soft palate and the posterior pharyngeal wall, with inability to breathe through the nose. A plastic operation was advised when the Wassermann reaction should become negative. After a course of antisyphilitic treatment, the Wassermann reaction was negative in September, 1924.

On Dec. 16, 1924, a plastic operation was performed by the use of endothermy, whereby an opening was made into the nasophaynx large enough to permit nasal breathing and decrease the difficulty in swallowing. An ordinary sewing needle, about 3 inches long, bent at an angle near the top and placed in a suitable handle, was used in making this dissection. The outline of the free border of the palate was determined by the use of a probe through the nose. The operation was painless and practically bloodless, and there was immediate relief of symptoms. A packing of vaseline gauze was left in the nasopharynx for 24 hours, after which no dressings or dilators were used. The post-operative reaction was slight, and there seemed to be little retraction or scarring of tissues.

On May 16, 1925, the patient still had good nasal breathing, but complained of some trouble in swallowing, and a second application ondothermia was made. At the present time the throat appears much as shown in the drawings, the last having been made Oct. 15, 1925.

DR. FREUDENTHAL: If endothermy is as simple and effective as Dr. Palmer mentioned, it ought to be an ideal method for treating certain kinds of adhesions. I have a case under observation, a very extensive adhesion between the septum and outer wall, following a small operation. At the slightest touch the patient bleeds terribly. I had to stop several times on account of such bleeding, and I wonder whether this method would be available in the case. If so, I would be very glad, indeed, to try it.

DR. PALMER: Endothermy is used principally in cancer. I do not know whether any cases have been reported where it was used in pharyngeal stenosis, but the chief difficulty encountered in the ordinary methods is bleeding, and while we had a slight amount of bleeding during this operation, I think it was due to faulty technique on my part. There was no post-operative hemorrhage.

DR. R. KRAMER (by invitation): In Paris I saw endothermy used in stenosis cases of the nose, nasopharynx and pharynx. It was a very simple ambulatory treatment, and the results were excellent.

Retromaxillary Abscess Following Tonsillectomy. Dr. Rudolph Kramer.

There has been a small number of cases of local and general infections reported following tonsillectomy. Most of the reported cases ended fatally. Personal communications have revealed a few cases not reported in the literature. These did not result in fatalities. It would be of great value if all such infections were reported, the causative factors investigated, and the treatment and the results of treatment recorded so that prophylactic and therapeutic measures could be definitely advocated. Two cases of suppuration following tonsillectomy have come under my care.

The first case was Mrs. G. S., age 22 years, who had had frequent attacks of tonsillitis. Shortly after the onset of an attack of acute tonsillitis, a tonsillectomy under local anesthesia was performed. An infection of the right side of the throat appeared after the operation and necessitated incision on two separate occasions. I saw her, for the first time, eight weeks after the tonsillectomy. There was trismus, swelling, pain and tenderness of the right parotid region. A fistula was present in the posterior portion of the right tonsillar fossa. There was swelling of the floor of the right external auditory canal. This was incised and greenish pus evacuated. There was a soft fluctuating tender swelling behind the right auricle. Hot moist applications were applied here for 24 hours. An incision was then made here and pus evacuated. A probe, passed through the retroauricular opening emerged from the incision in the external auditory canal. I was unable to pass a probe from the opening in the tonsillar fossa into the external canal. After two and one-half weeks of drainage and irrigation of the fistulae, the wounds healed up completely.

The second case is that of P. S., male, age 44 years, seen by me for the first time two and one-half weeks after a tonsillectomy had been performed in another city. Communication with the operator revealed that the patient had had a tonsillectomy performed under local anesthesia to remove a focus of infection. The pre-operative examination of his tonsils showed nothing noteworthy. The anesthesia was by means of pillar infiltration; the technic of tonsillectomy was the dissection and snare procedure; the post-operative course was smooth, the wounds in the throat being almost completely healed on the eleventh day. Twelve days post-operative, the patient traveled for about 24 hours by train. Towards the latter part of the trip he had pain in the left side of the throat. The following day the pain was more severe and the temperature was 100.° There appeared trismus, pain, swelling and tenderness of the left parotid region. When I saw him on the eighteenth day post-operative his temperature was 101;° the left parotid region was swollen and tender, and marked trismus was present. There was a small unhealed area in the left tonsillar fossa covered with a grayish exudate. The left pharyngeal wall was bulging, but not reddened. No evidence of suppuration was found. The condition remained stationary for two days. On the third day the temperature rose to 104;° there was pain and tenderness in the left side of the throat and the left tonsillar fossa wound was unhealed, red and sluggish in appearance. There was edema of the left side of the epipharynx and hypopharynx. The left parotid region was red and edematous. Under local anesthesia a 2½-inch horizontal incision was made below and parallel to the horizontal remus of the left mandible, extending as far back as the angle of the jaw. The facial vein and the sheath of the great vessel were retracted outwards. Small, slightly edematous lymph glands were encountered.

The deep fascia was incised and a Mayo scissors passed internal to the internal pterygoid muscle, where about 6 ounces of foul-smelling pus was found. The pus cavity ran up in the region of the posterior aspect of the tuberosity of the superior maxilla. Two drainage tubes were inserted. The wound healed up in two and one-half weeks, remained closed for two weeks and then an abscess formed and ruptured spontaneously, 18 hours after first giving symptoms of its appearance. There is now a narrow tract about 2½ inches long, extending upwards towards the region of the tonsillar fossa.

Both cases were done under local anesthesia; the technic employed was superficial injection of novocain into the tonsillar pillars. In the first patient the tonsillectomy was performed during the course of an acute tonsillitis and I am inclined to ascribe the resulting abscess to the presence of this acute infection at the time of operation. However, the path taken by the suppuration is rather peculiar, in that it led to the region of the auricle and external auditory canal. This coincides with the path followed by certain unusual forms of branchial fistulae, those of the first pharyngeal pouch. Were it not for the fact that it healed up completely, without radical removal, in so short a time, I would be in-

clined to class this case under the heading of branchial fistula of the first pharyngeal pouch.

The etiological factor in the second case is difficult to define. The preoperative condition was not, apparently, a factor. The operative procedure, as reported, does not seem likely to have caused a deep infection first appearing two weeks post-operative. The exposure on the twelfth day, with the throat unhealed, stands in direct chronological relationship to the appearance of symptoms indicating the onset of infection.

The infection in the first case never assumed threatening proportions; but in the second, the presence of hypopharyngeal edema indicated real danger and necessitated immediate external drainage of the abscess.

The treatment of these cases was incision and drainage. It may be necessary in cases such as the second one presented, to dissect deeply in the neck, going internal to the great vessels before pus is reached.

Some of the Newer Methods of X-ray Examination of the Paranasal Sinuses, etc. Dr. Harry A. Goalwin.

(Published in full in this issue.)

DISCUSSION.

DR. F. M. LAW: I was intensely interested in the presentation of the subject, and congratulate Dr. Goalwin on the clever and accurate method of measuring the optic foramen and the direction of the optic canal. I leave the discussion of the clinical value to the ophthalmologists and laryngologists, but there is one very important feature I would like to emphasize. After seeing these pictures and hearing Dr. Goalwin's presentation, you can all understand the necessity for personal contact with the Roentgenologist in studying these cases, rather than relying only upon the written report. The Roentgenologist may put down what he thinks is the trouble, but when you read it you may not read into it what he has in mind. Always have a personal consultation with the X-ray man, talk over the case with him, give him the benefit of your clinical knowledge and surgical experience, and together you can get a proper knowledge of what is present and what is best to be done.

DR. LE WALD: I would like to show a couple of lantern slides illustrating the question of the examination of the larynx and retropharyngeal space. This region is certainly one which lends itself readily to X-ray study, if one uses a little care in technique.

The first slide shows a very large adenoid that Dr. D. H. Jones took out at the Manhattan Eye, Ear, Nose and Throat Hospital. You see it narrows the space and makes a contrast shadow between the posterior wall of the nasophaynx and the air column in the nasopharynx. The next slide shows another adenoid case and a negative nasopharynx on the same slide for contrast. Dr. Kerley confirmed the *absence* of adenoid tissue in this latter case.

I take exception to what Dr. Goalwin said about the larynx. Much has been said about the difference between the male and female larynx. There is a difference in the ossification, but it does not vary greatly between the two sexes, according to Dr. Noback, Assistant Professor of Anatomy at New York University. We are still continuing this study among the students at Bellevue College. There is a variation in the ossification time in both sexes but it is not so definite that you can distinguish one sex from the other by the degree of ossification of the larynx. It is exceedingly important for the Roentgenologist to know the amount of ossification at a given age in order that he may not be let into an erroneous diagnosis of foreign body in the larynx. As Dr. Goalwin said, there is a definite center of ossification, just as in any of the bones of the body, such as we see in young subjects. This goes on in an orderly fashion, so that you can outline it at various ages in various subjects, but it does not take place at absolutely the same age in different individuals.

The question of the X-ray diagnosis of retropharyngeal abscess is of interest. This question has come up very recently at the Willard Parker Hospital.